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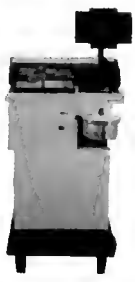

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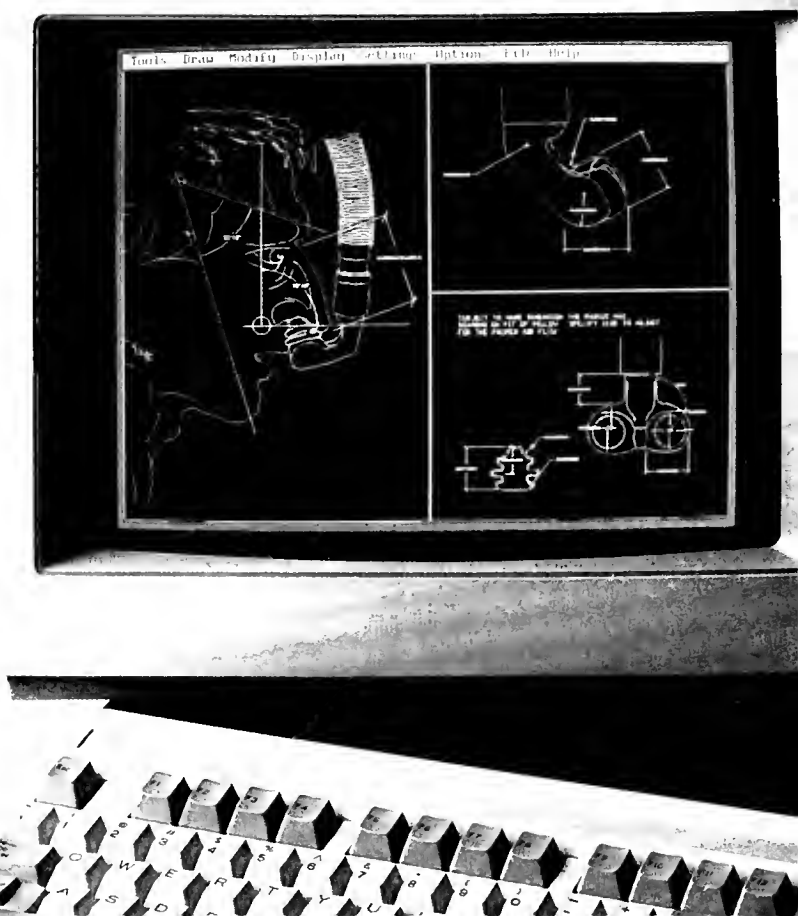

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RESPIRATORY CARE is indexed in *Hospital Literature Index* and in *Cumulative Index to Nursing* and *Allied Health Literature*.

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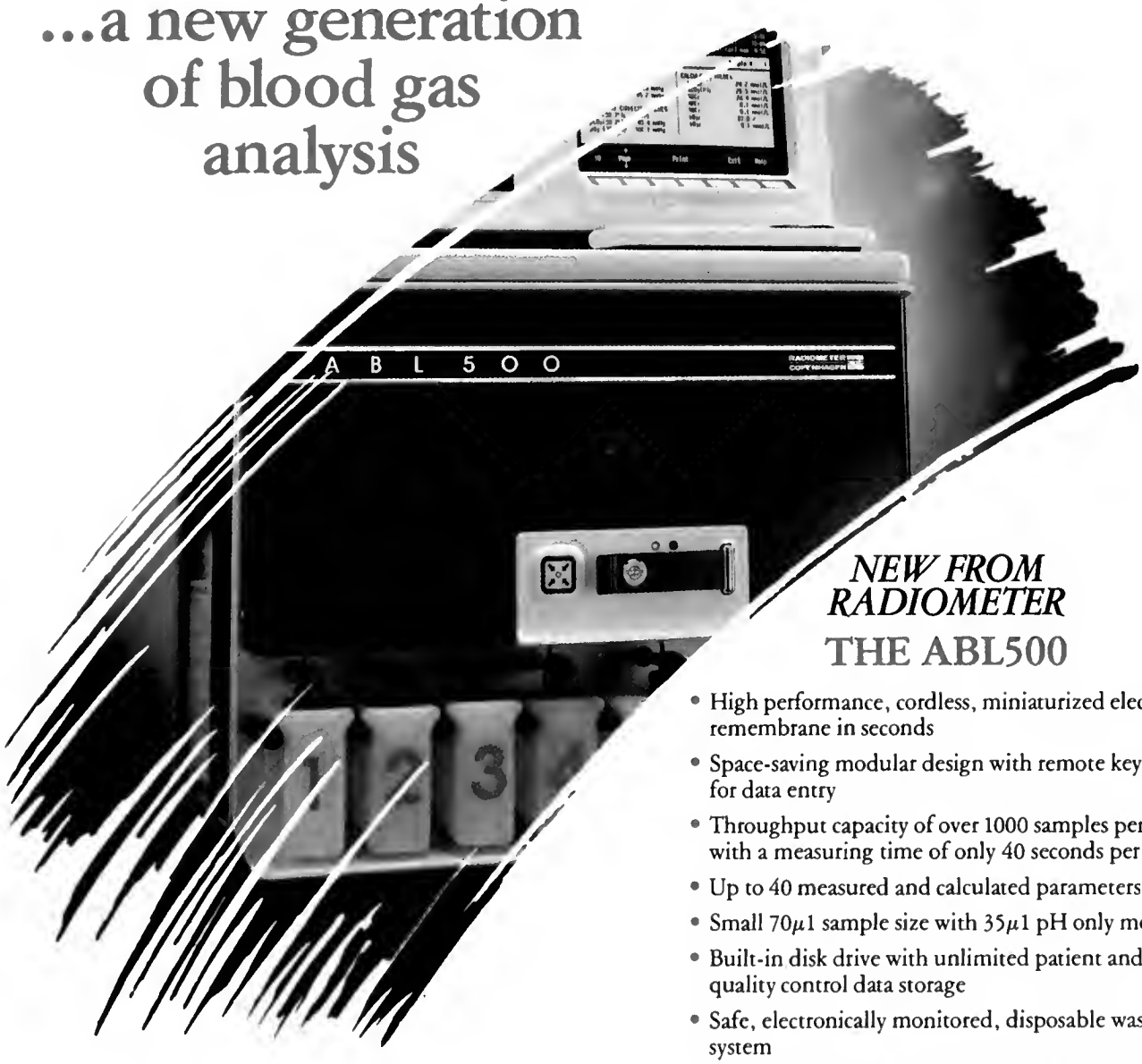

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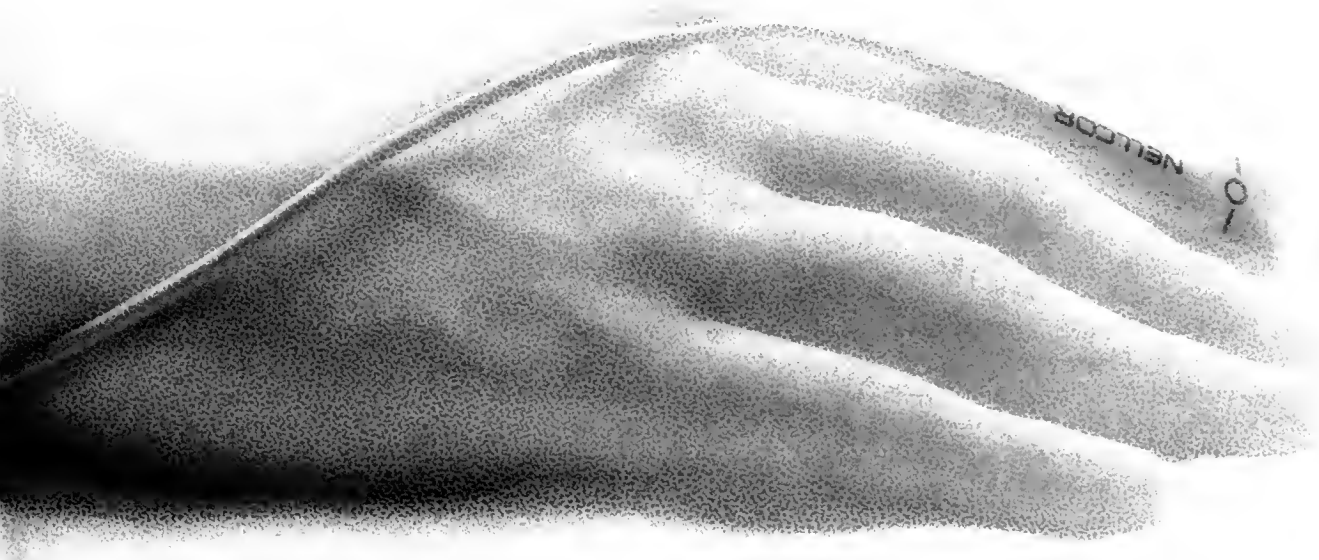

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Percutaneous Tracheostomy: A New Method—A Schachner, Y Ovil, J Sidi, M Rogev, Y Heilbronn, MJ Levy. Crit Care Med 1989;17: 1052.

A rapid method of definitive low tracheostomy utilizing an original instrument kit and percutaneous approach is described. Through a horizontal, shallow (1.5-cm) skin incision in the neck, a 12-ga needle is introduced into the tracheal lumen. A flexible metal guidewire is gently introduced through the needle, which is then removed. A specially designed percutaneous tracheostomy tool slides over the guidewire into the trachea; by squeezing its handles, the intercartilaginous space is enlarged, securing the placement of a cuffed tracheal cannula. After extensive and successful investigation in both cadaver and animal trials, the procedure was carried out in 80 patients who had a variety of underlying diseases, without serious complications. Of these 80 patients, 33 required airway control after neurosurgical interventions or after severe head trauma, 23 had percutaneous tracheostomy performed before radical excision for head and neck cancer, and the remaining 24 suffered from severe cardiorespiratory problems. Twenty-nine procedures were performed in the operating theater, and 51 procedures were carried out at the patient's bedside in the ICU, ED, or in the ward. There was no infection at the stoma site, and late healing was remarkable. It should be strongly emphasized that in the majority of our patients the procedure was carried out safely at the bedside. This resolved the logistic problem of moving very sick ICU patients (who are sometimes on high PEEP levels) to the OR. Although our experience is totally restricted to elective situations, we do postulate that it could be as effective in a variety of urgent situations.

On the Temporal Relationships Between Lung Function and Somatic Growth—DL Sherrill, A Camilli, MD Lebowitz. Am Rev Respir Dis 1989;140:638.

A sample population of healthy nonsmoking men (n = 416) and women (n = 608) subjects derived from all non-Hispanic white subjects 5 to 60 y of age enrolled in the Tucson Epidemiological Study of Airway Obstructive Disease was studied to evaluate the temporal relationships between various pulmonary function measures and somatic growth. Pulmonary function measures derived from the maximal expiratory flow volume (MEFV) curve included FVC, FEV_1, maximal expiratory flow after 50% of vital capacity had been expired ($\dot{V}_{max_{50}}$), and maximal midexpiratory flow ($FEF_{25-75\%}$). The lung function and somatic growth longitudinal data were characterized by a nonparametric polynomial smoothing spline model. This procedure yields an optimal fitted curve through the data, an estimate of the growth velocity curve, and 95% confidence bands. Temporal relationships between the fitted growth curves were determined examining the ages at which the growth velocity peaks (GVP) occur. In addition, the age of growth cessation was estimated using lower limits of the 95% confidence bands of each growth velocity curve. The results indicate that the point estimates of the somatic GVPs precede all peak lung function measurements derived from the MEFV curve in both men and women. The only statistically significant difference between when the GVPs for somatic growth and functional lung growth occurred was for FEV_1 and $FEF_{25-75\%}$ in men, though similar trends were apparent in all other variables, suggesting that the timing of maximal body growth velocity precedes that of maximal lung functional growth and that the early growth spurt in women includes the lungs. For the cessation of lung growth estimates, we found that the FEV_1 slope could not be distinguished from zero at 19.82 y of age for men and 17.88 y for women with similar values for FVC, $\dot{V}_{max_{50}}$, and $FEF_{25-75\%}$. Additional regression analysis was also applied to adult subjects to determine the age at which lung function increases its rate of decline; this was approximately 43 y of age for men and 37 y for women.

Submaximal Exercise in Emphysema and Malnutrition at Two Levels of Carbohydrate and Fat Intake—SA Goldstein, J Askanazi, DH Elwyn, et al. J Appl Physiol 1989; 67:1048.

Eight malnourished patients with emphysema (EMPH) and eight malnourished patients without evidence of lung disease (MALN) received an infusion of 5% dextrose plus electrolytes (D_5W) for 48 h and were then randomly assigned to a hypercaloric diet with either 53% of the calories as carbohydrate (CB) or with 55% as fat (FB) for the first week, maintaining a constant protein intake. The alternate diet was given the following week. Ventilation and gas exchange were measured during supine cycle ergometry at 0, 12, and 25 W during the D_5W, CB, and FB diet periods. At each exercise intensity, the EMPH group demonstrated a 12-15% greater O_2 consumption, a lower respiratory quotient, and an O_2 debt larger than that of the MALN group. Resting ventilation was higher during the CB than FB regimen in both groups of patients, but during the CB diet the EMPH group had a more exaggerated ventilatory response than the MALN group. The results demonstrate that EMPH patients have an unusual

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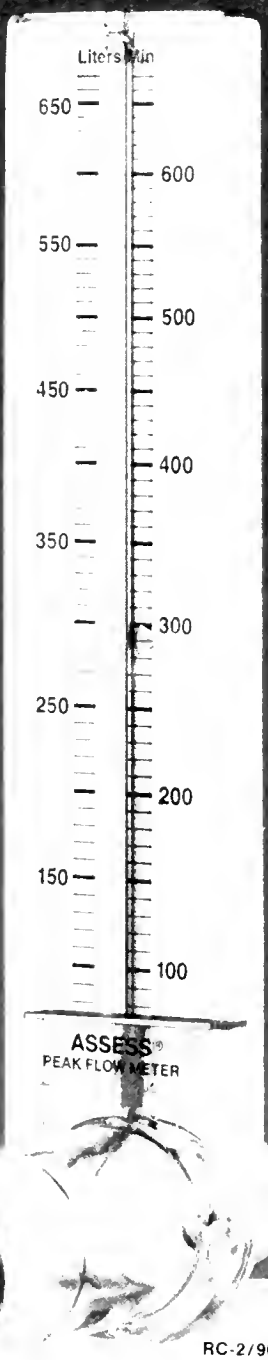

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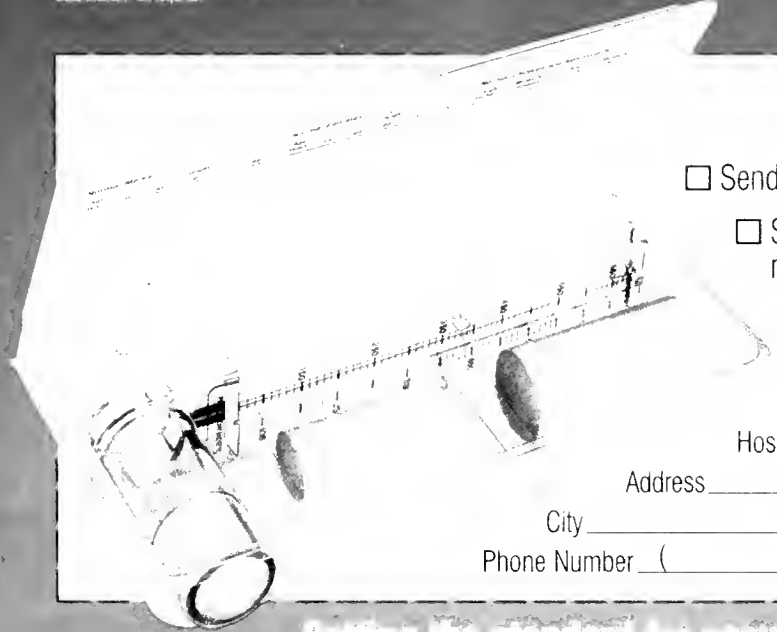

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metabolic pattern during hypercaloric feeding and exercise. Furthermore, in EMPH patients a FB regimen does not appear to create the additional stress on the respiratory system during exercise that is generated with a CB regimen.

Elevated Intracranial Pressure Increases Pulmonary Vascular Permeability to Protein—MD McClellan, IM Dauber, JV Weil. J Appl Physiol 1989;67:1185.

The syndrome of neurogenic pulmonary edema raises the question of whether there are neurological influences on pulmonary vascular permeability. Previous experimental models commonly produced severe hemodynamic alterations, complicating the distinction of increased permeability from increased hydrostatic forces in the formation of the pulmonary edema. Accordingly, we employed a milder central nervous system insult and measured the pulmonary vascular protein extravasation rate, which is a sensitive and specific indicator of altered protein permeability. After elevating intracranial pressure via cisternal saline infusion in anesthetized dogs, we used a dual isotope method to measure the protein leak index. This elevated intracranial pressure resulted in a nearly threefold rise in the protein leak index (54.1 $\pm$ 7.5 vs 20.2 $\pm$ 0.9). This central nervous system insult was associated with only mild increases in pulmonary arterial pressures and cardiac output. However, when we reproduced these hemodynamic changes with left atrial balloon inflation or isoproterenol infusion, we observed no effect on the protein leak index compared with control. Although the pulmonary arterial wedge pressure with intracranial pressure elevation remained $<$ 10 torr, increases in the extravascular lung water were demonstrated. The results suggest the existence of neurological influences on pulmonary vascular protein permeability. We conclude that neurological insults result in increased pulmonary vascular permeability to protein and subsequent edema formation, which could not be accounted for by hemodynamic changes alone.

Aminophylline Effects on Ventilatory Response to Hypoxia and Hyperoxia in Normal Adults—D Georgopoulos, SG Holtby, D Berezanski, NR Anthonisen. J Appl Physiol 1989;67:1150.

In 10 young normal adults, ventilation was evaluated with and without pretreatment with aminophylline, an adenosine blocker, while they breathed pure O_2 (1) after breathing room air and (2) after 25 min of isocapnic hypoxia (arterial O_2 saturation 80%). With and without aminophylline, 5 min of hyperoxia significantly increased inspiratory minute ventilation ($\dot{V}_I$) from the normoxic baseline. In control experiments, with hypoxia, $\dot{V}_I$ initially increased and then declined to levels that were slightly above the normoxic baseline. Pretreatment with aminophylline significantly attenuated the hypoxic ventilatory decline. During transitions to pure O_2 (cessation of carotid bodies' output), $\dot{V}_I$ and breathing patterns were analyzed breath by breath with a moving-average technique, searching for nadirs before and after hyperoxia. On placebo days, at the end of hypoxia, hyperoxia produced nadirs that were significantly lower than those observed with room-air breathing and also significantly lower than when hyperoxia followed normoxia, averaging, respectively, 6.41 $\pm$ 0.52, 8.07 $\pm$ 0.32, and 8.04 $\pm$ 0.39 (SE) L/min. This hypoxic depression was due to significant decrease in tidal volume and prolongation of expiratory time. Aminophylline partly prevented these alterations in breathing pattern; significant posthypoxic ventilatory depression was not observed. We conclude that aminophylline attenuated hypoxic central depression of ventilation, although it does not affect hyperoxic steady-state hyperventilation. Adenosine may play a modulatory role in hypoxic but not in hyperoxic ventilation.

Survival following Mechanical Ventilation for *Pneumocystis carinii* Pneumonia in Patients with the Acquired Immunodeficiency Syndrome: A Different Perspective—LS Efferen, D Nadarajah, DS Palat. Am J Med 1989;87:401.

Pulmonary infection is a frequent cause of morbidity and mortality in patients with acquired immunodeficiency syndrome (AIDS), and *Pneumocystis carinii* pneumonia (PCP) is the predominant infection in these patients. In those patients who experience progression to respiratory failure from PCP, the reported mortality rate has been between 87% to 100%. This, in addition to the ultimately fatal outcome of patients with AIDS, has led many physicians to question the advisability of instituting mechanical support for respiratory failure in the setting of PCP. It had been our impression that the outcome of patients on our service was not as poor as was generally reported. We therefore undertook a retrospective analysis of our clinical experience. We reviewed the clinical course of patients admitted to our service between December 1984 and June 1988 who required intubation and mechanical ventilation for PCP or presumed PCP. Thirty-three cases were identified with 18 survivors (54.5%) and 15 non-survivors (45.5%). Twenty-five of the 33 patients were intubated for their first episode of PCP, with 16 survivors (64%), whereas the remaining 8 patients were intubated for their second episode of PCP, with 2 survivors (25%). We were not able to identify any parameters that predicted survival, although the serum lactate

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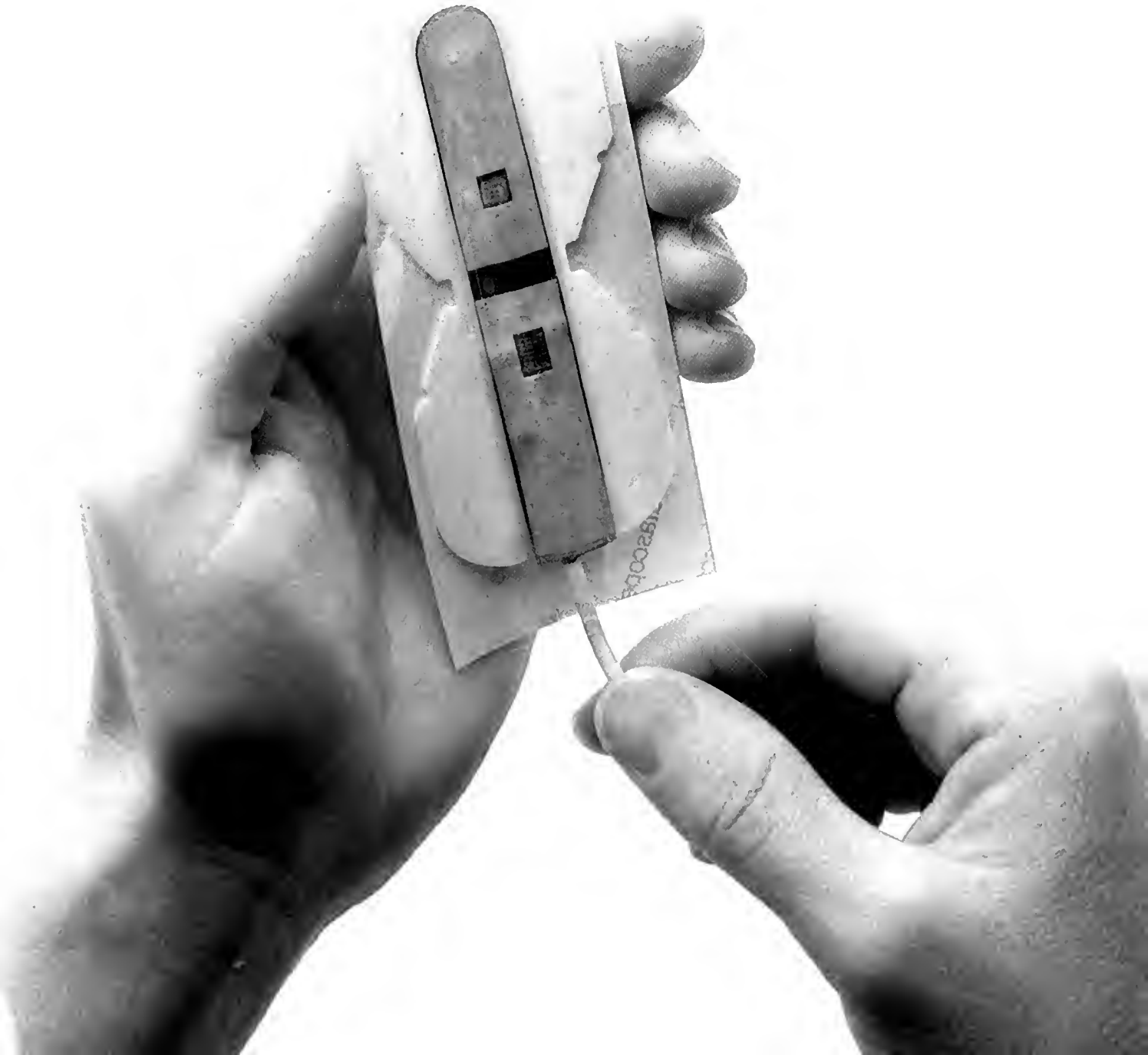

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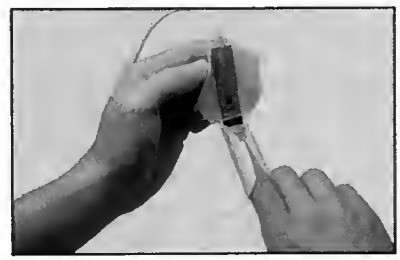

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dehydrogenase level was useful in following the response to treatment. It is our belief that there is a reasonable chance of survival for patients requiring mechanical ventilation for PCP. We question the wisdom of avoiding intubation and mechanical ventilation altogether in patients with PCP due to the presumption of fatality in this clinical situation.

Effect of Airway Pressure on Pericardial Pressure—MR Cabrera, GE Nakamura, DA Montague, RP Cole. Am Rev Respir Dis 1989;140:659.

The effect of applied positive airway pressure or PEEP on pericardial pressure was examined in closed-chest, anesthetized dogs with normal lungs and in animals with oleic acid-induced lung injury. Both groups were studied before and after vascular volume loading by dextran infusion. Pericardial pressure was measured using an air-filled flat balloon placed along the lateral left-ventricular free wall within the pericardial space. A linear relation between right-atrial pressure (RAP) and pericardial pressure (PP) was found (RAP = 3.9 $\pm$ 0.78 PP, r = 0.76, $P < 0.01$) that was independent of acute lung injury but was influenced by the application of PEEP. In the baseline volume condition, pericardial pressure increased linearly with an increase in PEEP to 15 cm H_2O. After volume loading, pericardial pressure increased fourfold, but, surprisingly, did not change with applied PEEP. These relations were similar in the two groups despite a reduction in total respiratory system compliance in the oleic acid group. This suggests that transmission of airway pressure to the pericardial surface is independent of the presence of acute lung injury and that changes in pericardial pressure in response to PEEP reflect both the transmission of airway pressure to the pericardial surface and, presumably, PEEP-related changes in cardiac volume and ventricular compliance.

Assessment of Patients' Experience of Discomforts during Respirator Therapy—I Bergbom-Engberg, H Haljamäe. Crit Care Med 1989;17: 1068.

Patients (n = 158) who had been respirator-treated and who could remember the treatment were retrospectively (after > 2 mo) interviewed about their experiences. Of all patients, 47% had felt anxiety and/or fear during the treatment. These feelings were intimately related to the experience of agony/panic ($p < 0.001$) and insecurity ($p < 0.001$). Inability to talk and communicate was found to be the dominating reason ($p < 0.001$) for evoking such feelings and also made it difficult for the patients to sleep and rest ($p < 0.05$); no correlation to pain was found. Difficulties to synchronize with the respirator in connection with suctioning also caused feelings of anxiety/fear ($p < 0.01$), agony/panic ($p < 0.01$), and insecurity ($p < 0.001$). Even as long as 4 y after respirator treatment, most patients (90%) who remember the treatment still recall the situation as unpleasant and stress-evoking. The isolation due to communication difficulties was a greater problem than direct airway-related nursing care activities. This relationship between communication difficulties and severe emotional reactions should be considered in the nursing care of respirator-treated patients.

Dexamethasone in the Treatment of Acute Mountain Sickness—BD Levine, K Yoshimura, T Kobayashi, M Fukushima, T Shibamoto, G Ueda. N Engl J Med 1989;321:1707.

Cerebral edema occurs in fatal cases of acute mountain sickness. Dexamethasone, commonly used to treat cerebral edema due to other causes, also reduces the symptoms of acute mountain sickness when given prophylactically. However, the efficacy of dexamethasone in the treatment of established acute mountain sickness remains uncertain. To investigate this question, we exposed six men in a hypobaric chamber to a simulated altitude of 3700 m (barometric pressure, 64 kPa [481 torr]) for 48 h on two occasions. Acute mountain sickness was diagnosed with use of a symptoms questionnaire, and dexamethasone (4 mg every 6 h) or placebo was then given in a randomized, double-blind, crossover fashion. Dexamethasone reduced the symptoms of acute mountain sickness by 63% ($p < 0.05$), whereas placebo had a minimal effect (reduction by 23%; p not significant). In spite of this response, one subject had mild cerebral edema on brain CT after both placebo and dexamethasone. Dexamethasone had no effect on fluid shifts, oxygenation, sleep apnea, urinary catecholamine levels, the appearance of chest radiographs or perfusion scans, serum electrolye levels, hematologic profiles, or the results of psychometric tests. Dexamethasone treatment was complicated by mild hyperglycemia in all subjects (mean [$\pm$ SE] glucose level, 7.3 $\pm$ 1.3 mmol/L [132 $\pm$ 23 mg/dL. We conclude that dexamethasone effectively reduces the symptoms of acute mountain sickness. However, it did not improve objective physiologic abnormalities related to exposure to high altitudes. We therefore recommend that dexamethasone be used only when descent is impossible, or to facilitate cooperation in evacuation efforts.

The Cost to the Central Nervous System of Climbing to Extremely High Altitude—TF Hornbein, BD Townes, RB Schoene, JR Sutton, CS Houston. N Engl J Med 1989;321: 1714.

To assess the possibility that climbing to extremely high altitude may result in hypoxic injury to the brain, we performed neuropsychological and physiologic testing on 35 mountaineers before and 1 to 30 days after ascent

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to altitudes between 5488 and 8848 m, and on 6 subjects before and after simulation in an altitude chamber of a 40-day ascent to 8848 m. Neuropsychological testing revealed a decline in visual long-term memory after ascent as compared with before; of 14 visual items of information on the Wechsler Memory Scale, fewer were recalled after ascent by both the simulated-ascent group (a mean [± SD] of 10.14 ± 1.68 items before, as compared with 7.00 ± 3.35 items after; $p < 0.05$) and the mountaineers (12.33 ± 1.96 as compared with 11.36 ± 1.88, $p < 0.05$). Verbal long-term memory was also affected, but only in the simulated-ascent group; of a total of 10 words, an average of 8.14 ± 1.86 were recalled before simulated ascent, but only 6.83 ± 1.47 afterward ($p < 0.05$). On the aphasia screening test, on which normal persons make an average of less than one error in verbal expression, the mountaineers made twice as many aphasic errors after ascent (1.03 ± .10) as before (0.52 ± 0.80; $p < 0.05$). A higher ventilatory response to hypoxia correlated with a reduction in verbal learning ($r = -0.88$, $p < 0.05$) and with poor long-term verbal memory ($r = -0.99$, $p < 0.01$) after ascent. An increase in the number of aphasic errors on the aphasia screening test also correlated with a higher ventilatory response to hypoxia in both the simulated-ascent group ($r = 0.94$, $p < 0.01$) and a subgroup of 11 mountaineers ($r = 0.59$, $p < 0.05$). We conclude that persons with more vigorous ventilatory response to hypoxia have more residual neurobehavioral impairment after returning to lower elevations. This finding may be explained by poorer oxygenation of the brain despite greater ventilation, perhaps because of a decrease in cerebral blood flow caused by hypocapnia that more than offsets the increase in arterial oxygen saturation.

Determinants and Limits of Pressure-Preset Ventilation: A Mathematical Model of Pressure Control—JJ Marini, PS Crooke III, JD Truwit. J Appl Physiol 1989;67: 1081.

In recent years, four square-wave modes of pressure-preset mechanical ventilation (PPV)—pressure control, pressure support, inverse ratio, and airway pressure release ventilation—have been introduced to clinical practice. Conceptually, they share important features. Yet, because there remains widespread uncertainty regarding their ventilatory characteristics, efficacy, and appropriate use, the potential range of application is only now being investigated. To construct a unifying mathematical model of PPV, we developed a system of equations for prediction of the major 'outcome' variables of PPV—tidal volume, minute ventilation, auto-positive end-expiratory pressure, mean alveolar pressure, and mechanical work—from the primary clinical 'inputs' from patient (resistance, compliance) and clinician (applied pressure, frequency, inspiratory time fraction). Our analysis revealed distinct bounding limits for the outcome variables of ventilation and pressure and important implications for their clinical determinants. Although simplifying assumptions were required to enable construction of this mathematical analogue of respiratory system behavior, this model provides a firm conceptual framework for understanding the physiological interactions between PPV and the patients they are intended to help.

Increased Chemoreceptor Output and Ventilatory Response to Sustained Hypoxia—D Georgopoulos, S Walker, NR Anthonisen. J Appl Physiol 1989;67:1157.

In adult humans the ventilatory response to sustained hypoxia (VRSH) is biphasic, characterized by an initial brisk increase, due to peripheral chemoreceptor (PC) stimulation, followed by a decline attributed to central depressant action of hypoxia. To study the effects of selective stimulation of PC on the ventilatory response pattern to hypoxia, the VRSH was evaluated after pretreatment with almitrine (A), a PC stimulant. Eight subjects were pretreated with A (75 mg po) or placebo (P) on 2 days in a single-blind manner. Two hours after drug administration, they breathed, in succession, room air (10 min), O_2 (5 min), room air (5 min), hypoxia [25 min, arterial O_2 saturation (S_{aO_2}) = 80%], O_2 (5 min), and room air (5 min). End-tidal CO_2 was kept constant at the normoxic baseline values. Inspiratory minute ventilation ($\dot{V}_I$) and breathing patterns were measured over the last 2 min of each period and during *minutes 3-5* of hypoxia, and nadirs in $\dot{V}_I$ were assessed just before and after O_2 exposure. Independent of the day, the VRSH was biphasic. With P and A pretreatment, early hypoxia increased $\dot{V}_I$ 4.6 ± 1 and 14.2 ± 1 (SE) L/min, respectively, from values obtained during the preceding room-air period. On A day the hypoxic ventilatory decline was significantly larger than that on P day, and on both days the decline was a constant fraction of the acute hypoxic response. On both days, at the end of hypoxia, exposure to O_2 resulted in a transient drop in $\dot{V}_I$ to values that were significantly lower than those observed when hyperoxia followed normoxia, indicating a central hypoxic depressant effect. This effect was more evident on A day. Our results suggest that, during sustained hypoxia, PC discharge is centrally modulated to result in ventilatory decline.

Regulation of Oxygen Concentration Delivered to Infants via Nasal Cannulas—NE Vain, LM Prudent, DP Stevens, MM Weeter, MJ Maisels. AJDC 1989;143;1458.

The administration of oxygen to infants via nasal cannulas is now a common

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PRESCRIBING INFORMATION

WARNING: RIBAVIRIN AEROSOL SHOULD NOT BE USED FOR INFANTS REQUIRING ASSISTED VENTILATION BECAUSE PRECIPITATION OF THE DRUG IN THE RESPIRATORY EQUIPMENT MAY INTERFERE WITH SAFE AND EFFECTIVE VENTILATION OF THE PATIENT. Conditions for safe use with a ventilator are still in development.

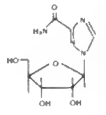
Deterioration of respiratory function has been associated with ribavirin use in infants, and in adults with chronic obstructive lung disease or asthma. Respiratory function should be carefully monitored during treatment. If initiation of ribavirin aerosol treatment appears to produce sudden deterioration of respiratory function, treatment should be stopped and re-instituted only with extreme caution and continuous monitoring.

Although ribavirin is not indicated in adults, the physician should be aware that it is teratogenic in animals (see CONTRAINDICATIONS).

DESCRIPTION:

Virazole® (ribavirin) Aerosol, an antiviral drug, is a sterile, lyophilized powder to be reconstituted for aerosol administration. Each 100 ml glass vial contains 6 grams of ribavirin, and when reconstituted to the recommended volume of 300 ml with sterile water for injection or sterile water for inhalation (no preservatives added), will contain 20 mg/ml ribavirin, pH approximately 5.5. Aerosolization is to be carried out in a SPAG-2 nebulizer only.

Ribavirin is 1-beta-D-ribofuranosyl-1,2,4-triazole-3-carboxamide, with the following structural formula:



Ribavirin, a synthetic nucleoside, is a stable, white, crystalline compound with a maximum solubility in water of 142 mg/ml at 25°C and with only a slight solubility in ethanol. The empirical formula is $C_8H_{12}N_4O_5$ and the molecular weight is 244.2 Daltons.

CLINICAL PHARMACOLOGY:

Antiviral effects:

Ribavirin has antiviral inhibitory activity *in vitro* against respiratory syncytial virus,[1] influenza virus, and herpes simplex virus. Ribavirin is also active against respiratory syncytial virus (RSV) in experimentally infected cotton rats.[2]

In cell cultures, the inhibitory activity of ribavirin for RSV is selective. The mechanism of action is unknown. Reversal of the *in vitro* antiviral activity by guanosine or xanthosine suggests ribavirin may act as an analogue of these cellular metabolites.

Immunologic effects:

Neutralizing antibody responses to RSV were decreased in ribavirin treated compared to placebo treated infants.[3] The clinical significance of this observation is unknown. In rats, ribavirin resulted in lymphoid atrophy of thymus, spleen, and lymph nodes. Humoral immunity was reduced in guinea pigs and ferrets. Cellular immunity was also mildly depressed in animal studies.

Microbiology:

Several clinical isolates of RSV were evaluated for ribavirin susceptibility by plaque reduction in tissue culture. Plaques were reduced 85-98% by 16 μg/ml, however, plaque reduction varies with the test system. The clinical significance of these data is unknown.

Pharmacokinetics:

Assay for ribavirin in human materials is by a radioimmunoassay which detects ribavirin and at least one metabolite.

Ribavirin administrered by aerosol is absorbed systemically. Four pediatric patients inhaling ribavirin aerosol administered by face mask for 2.5 hours each day for 3 days had plasma concentrations ranging from 0.44 to 1.55 μM, with a mean concentration of 0.76 μM. The plasma half-life was reported to be 9.5 hours. Three pediatric patients inhaling ribavirin aerosol administered by face mask or mist tent for 20 hours each day for 5 days had plasma concentrations ranging from 1.5 to 14.3 μM with a mean concentration of 6.8 μM.

It is likely that the concentration of ribavirin in respiratory tract secretions is much higher than plasma concentrations in view of the route of administration.

The bioavailability of ribavirin aerosol is unknown and may depend on the mode of aerosol delivery. After aerosol treatment, peak plasma concentrations are less than the concentration that reduced RSV plaque formation in tissue culture by 85 to 98%. After aerosol treatment, respiratory tract secretions are likely to contain ribavirin in concentrations many fold higher than those required to reduce plaque formation. However, RSV is an intracellular virus and serum concentrations may better reflect intracellular concentrations in the respiratory tract than respiratory secretion concentrations.

In man, rats, and rhesus monkeys, accumulation of ribavirin and/or metabolites in the red blood cells has been noted, plateauing in red cells in man in about 4 days and gradually declining with an apparent half-life of 40 days. The extent of accumulation of ribavirin following inhalation therapy is not well defined.

INDICATIONS AND USAGE:

Ribavirin aerosol is indicated in the treatment of carefully selected hospitalized infants and young children with severe lower respiratory tract infections due to respiratory syncytial virus (RSV). In two placebo-controlled trials in infants hospitalized with RSV lower respiratory tract infection, ribavirin aerosol treatment had a therapeutic effect, as judged by the reduction by treatment day 3 of severity of clinical manifestations of disease.[3,4] Virus titers in respiratory secretions were also significantly reduced with ribavirin in one of these studies.[4]

Only severe RSV lower respiratory tract infection is to be treated with ribavirin aerosol. The vast majority of infants and children with RSV infection have no lower respiratory tract disease or have disease that is mild, self-limited, and does not require hospitalization or antiviral treatment. Many children with mild lower respiratory tract involvement will require shorter hospitalization than would be required for a full course of ribavirin aerosol (3 to 7 days) and should not be treated with the drug. Thus the decision to treat with ribavirin aerosol should be based on the severity of the RSV infection.

The presence of an underlying condition such as prematurity or cardiopulmonary disease may increase the severity of the infection and its risk to the patient. High risk infants and young children with these underlying conditions may benefit from ribavirin treatment, although efficacy has been evaluated in only a small number of such patients.

Ribavirin aerosol treatment must be accompanied by and does not replace standard supportive respiratory and fluid management for infants and children with severe respiratory tract infection.

Diagnosis:

RSV infection should be documented by a rapid diagnostic method such as demonstration of viral antigen in respiratory tract secretions by immunofluorescence[3,4] or ELISA[5] before or during the first 24 hours of treatment. Ribavirin aerosol is indicated only for lower respiratory tract infection due to RSV. Treatment may be initiated while awaiting rapid diagnostic test results. However, treatment should not be continued without documentation of RSV infection.

CONTRAINDICATIONS:

Ribavirin is contraindicated in women or girls who are or may become pregnant during exposure to the drug. Ribavirin may cause fetal harm and respiratory syncytial virus infection is self-limited in this population. Ribavirin is not completely cleared from human blood even four weeks after administration. Although there are no pertinent human data, ribavirin has been found to be teratogenic and/or embryolethal in nearly all species in which it has been tested. Teratogenicity was evident after a single oral dose of 2.5 mg/kg in the hamster and after daily oral doses of 10 mg/kg in the rat. Malformations of skull, palate, eye, jaw, skeleton, and gastrointestinal tract were noted in animal studies. Survival of fetuses and offspring was reduced. The drug causes embryolethality in the rabbit at daily oral dose levels as low as 1 mg/kg.

WARNINGS:

Ribavirin administered by aerosol produced cardiac lesions in mice and rats after 30 and 36 mg/kg, respectively, for 4 weeks, and after oral administration in monkeys at 120 and rats at 154 to 200 mg/kg for 1 to 6 months. Ribavirin aerosol administered to developing ferrets at 60 mg/kg for 10 or 30 days resulted in inflammatory and possible emphysematous changes in the lungs. Proliferative changes were seen at 131 mg/kg for 30 days. The significance of these findings to human administration is unknown.

Ribavirin lyophilized in 6 gram vials is intended for use as an aerosol only.

PRECAUTIONS:

General:

Patients with lower respiratory tract infection due to respiratory syncytial virus require optimum monitoring and attention to respiratory and fluid status.

Drug Interactions:

Interactions of ribavirin with other drugs such as digoxin, bronchodilators, other antiviral agents, antibiotics, or antimetabolites has not been evaluated. Interference by ribavirin with laboratory tests has not been evaluated.

Carcinogenesis, mutagenesis, impairment of fertility:

Ribavirin induces cell transformation in an *in vitro* mammalian system (Balb/C3T3 cell line). However, *in vivo* carcinogenicity studies are incomplete. Results thus far, though inconclusive, suggest that chronic feeding of ribavirin to rats at dose levels in the range of 16-60 mg/kg body weight can induce benign mammary, pancreatic, pituitary and adrenal tumors.

Ribavirin is mutagenic to mammalian (L5178Y) cells in culture. Results of microbial mutagenicity assays and a dominant lethal assay (mouse) were negative.

Ribavirin causes testicular lesions (tubular atrophy) in adult rats at oral dose levels as low as 16 mg/kg/day (lower doses not tested), but fertility of ribavirin-treated animals (male or female) has not been adequately investigated.

Pregnancy:

Teratogenic Effects: Pregnancy Category X. See "Contraindications" section.

Nursing Mothers: Use of ribavirin aerosol in nursing mothers is not indicated because RSV infection is self-limited in this population. Ribavirin is toxic to lactating animals and their offspring. It is not known whether the drug is excreted in human milk.

ADVERSE REACTIONS:

Approximately 200 patients have been treated with ribavirin aerosol in controlled or uncontrolled clinical studies.

Pulmonary function significantly deteriorated during ribavirin aerosol treatment in six of six adults with chronic obstructive lung disease and in four of six asthmatic adults. Dyspnea and chest soreness were also reported in the latter group. Minor abnormalities in pulmonary function were also seen in healthy adult volunteers.

Several serious adverse events occurred in severely ill infants with life-threatening underlying diseases, many of whom required assisted ventilation. The role of ribavirin aerosol in these events is indeterminate. The following events were associated with ribavirin use:

Pulmonary: Worsening of respiratory status, bacterial pneumonia, pneumothorax, apnea, and ventilator dependence.

Cardiovascular: Cardiac arrest, hypotension, and digitalis toxicity.

There were 7 deaths during or shortly after treatment with ribavirin aerosol. No death was attributed to ribavirin aerosol by the investigators.

Some subjects requiring assisted ventilation have experienced serious difficulties, which may jeopardize adequate ventilation and gas exchange. Precipitation of drug within the ventilatory apparatus, including the endotracheal tube, has resulted in increased positive end expiratory pressure and increased positive inspiratory pressure. Accumulation of fluid in tubing ("rain out") has also been noted.

Although anemia has not been reported with use of the aerosol, it occurs frequently with oral and intravenous ribavirin, and most infants treated with the aerosol have not been evaluated 1 to 2 weeks post-treatment when anemia is likely to occur. Reticulocytosis has been reported with aerosol use.

Rash and conjunctivitis have been associated with the use of ribavirin aerosol.

Overdosage:

No overdosage with ribavirin by aerosol administration has been reported in the human. The LD_{50} in mice is 2 gm orally. Hypoactivity and gastrointestinal symptoms occurred. In man, ribavirin is sequestered in red blood cells for weeks after dosing.

DOSAGE AND ADMINISTRATION

Before use, read thoroughly the Viratek Small Particle Aerosol Generator (SPAG) Model SPAG-2 Operator's Manual for small particle aerosol generator operating instructions.

Treatment was effective when instituted within the first 3 days of respiratory syncytial virus lower respiratory tract infection.[3] Treatment early in the course of severe lower respiratory tract infection may be necessary to achieve efficacy.

Treatment is carried out for 12-18 hours per day for at least 3 and no more than 7 days, and is part of a total treatment program. The aerosol is delivered to an infant oxygen hood from the SPAG-2 aerosol generator. Administration by face mask or oxygen tent may be necessary if a hood cannot be employed (see SPAG-2 manual). However, the volume of distribution and condensation area are larger in a tent and efficacy of this method of administering the drug has been evaluated in only a small number of patients. Ribavirin aerosol is not to be administered with any other aerosol generating device or together with other aerosolized medications. Ribavirin aerosol should not be used for patients requiring simultaneous assisted ventilation (see Boxed Warnings).

Virazole is supplied as 6 grams of lyophilized drug per 100 ml vial for aerosol administration only. By sterile technique, solubilize drug with sterile USP water for injection or inhalation in the 100 ml vial. Transfer to the clean, sterilized 500 ml widemouth Erlenmeyer flask (SPAG-2 Reservoir) and further dilute to a final volume of 300 ml with sterile USP water for injection or inhalation. The final concentration should be 20 mg/ml. **Important:** This water should not have had any antimicrobial agent or other substance added. The solution should be inspected visually for particulate matter and discoloration prior to administration. Solutions that have been placed in the SPAG-2 unit should be discarded at least every 24 hours and when the liquid level is low before adding newly reconstituted solution.

Using the recommended drug concentration of 20 mg/ml ribavirin as the starting solution in the drug reservoir of the SPAG unit, the average aerosol concentration for a 12 hour period would be 190 micrograms/liter (0.19 mg/l) of air.

HOW SUPPLIED:

Virazole® (ribavirin) Aerosol is supplied in 100 ml glass vials with 6 grams of sterile, lyophilized drug which is to be reconstituted with 300 ml sterile water for injection or sterile water for inhalation (no preservatives added) and administered only by a small particle aerosol generator (SPAG-2). Vials containing the lyophilized drug powder should be stored in a dry place at 15-25°C (59-78°F). Reconstituted solutions may be stored, under sterile conditions, at room temperature (20-30°C, 68-86°F) for 24 hours. Solutions which have been placed in the SPAG-2 unit should be discarded at least every 24 hours.

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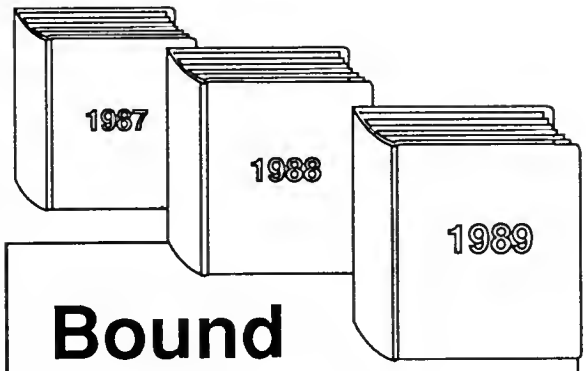

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practice in neonatal units, although the inspired oxygen concentration reaching the patient's airway is unknown. We measured the hypopharyngeal oxygen concentration in 10 infants who were receiving oxygen via nasal cannulas and assessed the impact of changes in the flowrate and inspired oxygen concentration. Weaning these infants by reducing the flowrate, even if changes are slight, produces clinically important changes in the oxygen concentration reaching the airway. Such changes are poorly tolerated by infants with chronic lung disease. Changing the flowrate and inspired oxygen concentration, rather than the flowrate alone, provides greater precision and is likely to avoid excessive and abrupt changes in the oxygen concentration reaching the airway.

The Effects of Cigarette Smoking and Smoking Cessation on the Carbon Monoxide Diffusing Capacity of the Lung in Asymptomatic Subjects—RJ Knudson, WT Kaltenborn, B Burrows. Am Rev Respir Dis 1989;140:645.

The single-breath carbon monoxide diffusing capacity (D_L) was measured together with standard spirometry as part of a survey of a randomly selected community population sample. Based on answers to a self-administered questionnaire, subjects free of respiratory symptoms or disease were identified. Data from subjects who had never regularly smoked cigarettes had been used to derive reference equations for the test variables, and data from the remaining subjects who had smoked were examined to determine the effect of smoking and smoking cessation on the D_L. From this cross-sectional analysis, it is apparent that cigarette smoking is associated with a decrease in D_L that occurs very soon after beginning the cigarette habit. There is an irreversible decrease in D_L with cumulative cigarette consumption, but also a reversible phenomenon that leads to rapid improvement in D_L on smoking cessation.

Lung Pressure and Gas Transport During High-Frequency Airway and Chest Wall Oscillation—MCK Khoo, T Ye, NH Tran. J Appl Physiol 1989; 67:985.

The major goal of this study was to compare gas exchange, tidal volume (V_T), and dynamic lung pressures resulting form high-frequency airway oscillation (HFAO) with the corresponding effects in high-frequency chest wall oscillation (HFCWO). Eight anesthetized paralyzed dogs were maintained eucapnic with HFAO and HFCWO at frequencies ranging from 1 to 16 Hz in the former and 0.5 to 8 Hz in the latter. Tracheal (ΔPtr) and esophageal (ΔPes) pressure swings, V_T, and arterial blood gases were measured in addition to respiratory impedance and static pressure-volume curves. Mean positive pressure (25-30 cm H_2O) in the chest cuff associated with HFCWO generation decreased lung volume by ~200 mL and increased pulmonary impedance significantly. Aside from this decrease in functional residual capacity (FRC), no change in lung volume occurred as a result of dynamic factors during the course of HFCWO application. With HFAO, a small degree of hyperinflation occurred only at 16 Hz. Arterial P_{O_2} decreased by 5 torr on average during HFCWO. V_T decreased with increasing frequency in both cases, but V_T during HFCWO was smaller over the range of frequencies compared with HFAO. ΔPes and ΔPtr between 1 and 8 Hz were lower than the corresponding pressure swings obtained with conventional mechanical ventilation (CMV) applied at 0.25 Hz. ΔPes was minimized at 1 Hz during HFCWO; however, ΔPtr decreased continuously with decreasing frequency and below 2 Hz became progressively smaller than the corresponding values obtained with HFAO and CMV.

Measurement of Pharyngeal Volume by Digitized Magnetic Resonance Imaging: Effect of Nasal Continuous Positive Airway Pressure—NC Abbey, AJ Block, D Green, A Mancusco, DW Hellard. Am Rev Respir Dis 1989;140:717.

Pharyngeal size is thought to play an important role in the pathogenesis of snoring and obstructive sleep apnea. It has been hypothesized that nasal continuous positive airway pressure (CPAP) works by enlarging pharyngeal size and splinting the airway open. In this study, we selected 12 heavy snorers and abolished their snoring with nasal CPAP in our sleep laboratory. Using magnetic resonance imaging and a computer program utilizing a digitizing pad, we measured these awake subjects' pharyngeal volumes without and with the nasal CPAP apparatus on at the level used to abolish their snoring. We found an average 27.7% increase in pharyngeal volume with nasal CPAP. We have visually shown an increase in pharyngeal size with the use of nasal CPAP in a cohort of heavy snorers.

Expiratory Glottic Widening in Asthmatic Subjects during Exercise-induced Bronchoconstriction—I Rubinstein, N Zamel, AS Slutsky, AS Rebuck, and V Hoffstein. Am Rev Respir Dis 1989;140:606.

Glottic caliber is modulated in a way that optimizes the airway resistance and the work of breathing. The midexpiratory glottic narrowing that normally occurs during tidal breathing is enhanced during histamine- or methacholine-induced airway narrowing. Exercise is associated with increased ventilatory demand and midexpiratory glottic widening in healthy subjects. However, the relative contribution of glottic aperture to airflow limitation during exercise-induced asthma is unknown. Conse-

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quently, we used the acoustic reflection technique to test whether a decrease in midexpiratory glottic size occurred also during exercise-induced bronchoconstriction in seven asthmatic subjects. Exercise resulted in a 38 ± 16% (SD) fall in FEV_1 from the baseline value ($p < 0.001$) and a significant increase in midexpiratory glottic cross-sectional area from 1.89 ± 0.45 cm^2 before to 2.36 ± 0.71 cm^2 during bronchoconstriction ($p < 0.05$). This midexpiratory glottic widening was also associated with increased tidal flow rates. We conclude that in marked contrast to airway obstruction induced by histamine or methacholine, exercise-induced asthma is associated with midexpiratory glottic widening, which occurs concomitantly with a decrease in lower airway caliber. We suggest that modulation of glottic aperture during acute asthma is heterogenous and depends partly on the initiating stimulus.

The Effect of Histamine Challenge on Platelet Count and Platelet Activation—CJ Duggan, WD Castle, MD Parmentier, N Berend. Aust NZ J Med 1989;19:327.

There is conflicting evidence of platelet involvement in asthmatic bronchoconstriction. We sought direct evidence of platelet activation and indirect evidence of platelet sequestration following histamine challenge (HC) in 14 asthmatics and seven nonasthmatic control subjects. Peripheral platelet counts and levels of β thromboglobulin (BTG) were measured immediately before and following HC in the 21 subjects. There were no significant differences in the platelet counts or BTG levels before or after HC, within or between the two groups. These results do not support platelet activation or accumulation in the lungs occurring immediately following histamine-induced bronchoconstriction in asthmatics.

Cause and Evaluation of Chronic Dyspnea in a Pulmonary Disease Clinic—MR Pratter, FJ Curley, J Dubois, RS Irwin. Arch Intern Med 1989;149:2277.

To test whether, in patients with chronic dyspnea, a diagnostic approach based on objective confirmation of suspected diagnoses would be superior to one based on clinical impression alone, we prospectively studied 85 patients with a primary complaint of dyspnea seen in a pulmonary subspecialty clinic. We achieved 100% success in determining the causes of dyspnea compared with only 66% accuracy based on clinical impression alone. Four groups of disorders, asthma, chronic obstructive pulmonary disease, interstitial lung diseases, and cardiomyopathy accounted for two thirds of the cases. Findings on the history and physical examination were too nonspecific to determine the specific diagnosis. Pulmonary function testing including a methacholine bronchoprovocation challenge, were the most useful diagnostic test, particularly for chronic obstructive pulmonary disease and asthma. Chest roentgenogram was most useful for interstitial lung disease, and comprehensive exercise testing for dyspnea due to psychogenic factors or deconditioning. Specific therapy was effective in reducing or eliminating dyspnea in the majority of cases. We conclude that a diagnostic approach to chronic dyspnea based on objective findings and verification, rather than clinical impression alone, will consistently lead to an accurate diagnosis and an improved therapeutic outcome.

Morphology of the Uvula in Obstructive Sleep Apnea—JL Stauffer, MK Buick, EO Bixler, et al. Am Rev Respir Dis 1989;140:724.

Alterations in pharyngeal structure and function are considered fundamental in the pathogenesis of obstructive sleep apnea (OSA). However, little is known about morphologic features of the pharynx in patients with OSA. We therefore studied the tissue composition of the uvula (midsagittal section) in patients with OSA, using a quantitative, morphometric point-counting technique. Uvula tissue was obtained by uvulopalatopharyngoplasty (UPPP) in 33 patients (mean number of apneas per hour of sleep = 32.7 ± 52) and by autopsy in 22 normal subjects not known to have OSA. All statistical comparisons were controlled for differences caused by age and body mass index. Patients with OSA had a significantly greater percentage of muscle in the uvula (18.1 ± 1.9% versus 9.3 ± 2.1%, $p = 0.02$) than did normal subjects. A significant difference in fat content was also found (9.5 ± 1.4% in patients versus 4.0 ± 1.0% in normal subjects, $p < 0.02$). These differences between patients with OSA and control subjects could not be accounted for by anthropometric or sex differences. The percentage of uvula fat tissue was significantly related to the frequency of apneas and hypopneas in sleep ($r = 0.43$, $p < 0.01$). Uvula morphology in 6 nonapneic snorers undergoing UPPP was similar to that of patients with OSA. We conclude that the uvula in patients with OSA contains more muscle and fat than the uvula in control subjcts, possibly contributing to pharyngeal narrowing in OSA.

Respiratory Risk Factors and Mortality: Longitudinal Studies in Washington County, Maryland—MS Tockman and GW Comstock. Am Rev Respir Dis 1989;140:S56.

Risk factors for temporal changes in chronic respiratory disease mortality were evaluated from two studies conducted in Washington County, Maryland. The first examined the mortality of a private census population (> 35,000 whites) enumerated in

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1963 over two subsequent time periods by age, sex, and initial smoking status. The second examined the 10-y mortality of a subset of the 1963 census (884 men who had undergone spirometry). We observed a fall in age-adjusted mortality from all causes and from arteriosclerotic heart disease (ASHD), but an increase in COPD mortality. However, the increase in these chronic pulmonary deaths is essentially confined to persons who were smoking cigarettes at the beginning of the study period. Furthermore, while smokers showed an increased mortality risk for all causes, the excess mortality risk did not fall uniformly across cigarette smokers. It is a major observation of this study that all-cause and cardiovascular (as well as pulmonary) mortality are significantly more often found among subjects with ventilatory impairment (independent of smoking status). Reasons for the association of ASHD mortality with impaired forced expiration are discussed. Thus, men at increased risk for three (ASHD, lung cancer, COPD) of the five leading causes of death (three of eight for women) may be identified by spirometry. Perhaps it is time that this test was more generally applied.

Hypoxic Ventilatory Response and Arterial Desaturation During Heavy Work—SR Hopkins, DC McKenzie. J Appl Physiol 1989;67:1119.

Arterial desaturation in athletes during intense exercise has been reported by several authors, yet the etiology of this phenomenon remains obscure. Inadequate pulmonary ventilation, due to a blunted respiratory drive, has been implicated as a factor. To investigate the relationship between the ventilatory response to hypoxia, exercise ventilation, and arterial desaturation, 12 healthy male subjects [age, 23.8 ± 3.6 y; height 181.6 ± 5.6 cm; weight 73.7 ± 6.2 kg; and maximal O_2 uptake ($\dot{V}O_{2max}$ 63.0 ± 2.2 ml · kg $^{-1}$ min^{-1})] performed a 5-min treadmill test at 100% of $\dot{V}O_{2max}$, during which arterial blood samples and ventilatory data were collected every 15 s. Alveolar P_{O_2} (P_{AO_2}) was determined using the ideal gas equation. On a separate occasion the ventilatory response to isocapnic hypoxia was measured. Arterial P_{O_2} decreased by an average of 29 torr during the test, associated with arterial desaturation [arterial O_2 saturation (S_{aO_2}) 92.0%]. P_{AO_2} was maintained; however, alveolar-arterial gas pressure difference increased progressively to > 40 torr. Minimal hypocapnia was observed, despite marked metabolic acidosis. There was no significant correlation observed between hypoxic drives and ventilation-to-O_2 uptake ratio or S_{aO_2} ($r = 0.1$ and 0.06, respectively, p = NS). These data support the conclusions that hypoxic drives are not related to maximal exercise ventilation or to the development of arterial desaturation during maximal exercise.

Prognosis of Chronic Obstructive Pulmonary Disease: The Dutch Experience—DS Postma and HJ Sluiter. Am Rev Respir Dis 1989; 140:S100.

This article summarizes the Dutch experience with prognosis in three groups of patients with chronic obstructive pulmonary disease. (1) 129 patients with severe chronic airflow obstruction (mean FEV_1 610 mL, range 350 to 910 mL); (2) 138 patients with less severe airflow obstruction (mean FEV_1 1,880 mL, range 1,200 to 2,500 mL); (3) 81 patients out of group 2 with decline in FEV_1 over time, without oral corticosteroids. It is clear that, at least in the presence of regular therapy, next to cessation of smoking, a higher reversibility of airflow obstruction and lower degree of airway hyperreactivity are important predictors of a slower decline in FEV_1. In our opinion these findings point to the urgency for clinicians to change the nihilistic attitude towards patients with CAO, in that both smoking cessation and an early therapy directed at the reversible part of airflow obstruction and airway hyperreactivity may be able to prevent deterioration. Future studies have to show whether long-term, ongoing therapy with corticosteroids may even change the progression and survivial in far advanced disease.

Breathing During Sleep With Mild Hypoxia—C Kazuo, M Ohi, M Hirai, T Kuriyama, Y Sagawa, and K Kuno. J Appl Physiol 1989;67:1198.

To investigate ventilatory response to mild hypoxia during nonrapid-eye-movement sleep, we administered ~16% O_2 (which corresponds to concentrations found in commercial high altitude aircraft) to 12 normal subjects by using an air entrainment mask, which did not alter the breathing pattern during this study. Under mild hypoxia, inspiratory minute ventilation during sleep showed an initial rapid increase ($p < 0.001$) but then declined significantly ($p < 0.001$) and stabilized. Stable levels differed among individuals and, compared with those measured before hypoxia, were significantly lower in some subjects, higher in one, and essentially unchanged in the others. The initial rapid increase in minute ventilation after mild hypoxia during sleep correlated with the respective values of hypoxic ventilatory response during the awake state ($p < 0.01$), but the final lowered levels did not. We conclude that the ventilatory response after mild hypoxia during sleep is biphasic and hypoxic depression exerts considerable influence on ventilation under mild hypoxia during sleep. So we should take hypoxic depression into consideration to evaluate the response to hypoxia during sleep.

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The Cleveland Clinic's Initial Experience with Transtracheal Oxygen Therapy

James P Adamo MD, Atul C Mehta MD, Kay Stelmach RN RRT, David Meeker MD, Thomas Rice MD, and James K Stoller MD

The advantages of improved efficiency of oxygen delivery—enhanced patient compliance and improved patient functional status—recommend transtracheal oxygen therapy over oxygen delivery by nasal cannulae. However, complications of transtracheal oxygen therapy also have been described. We report our initial experience with 21 patients undergoing transtracheal oxygen therapy (TTOT) at the Cleveland Clinic between October 1986 and June 1988. Overall, a total of 120 adverse events grouped into 25 types and 4 general categories are noted. On average, each patient experienced 5.7 complications or 0.7 complications per patient-month. Overall, few complications were severe enough to discourage continued use of TTOT; only 4 patients discontinued TTOT as a result of problems they encountered. No deaths resulted from TTOT, although a few serious and potentially serious complications occurred. These included acute respiratory failure, misplacement of catheters into the middle and anterior mediastinum, and moderate hemoptysis. Most other complications were minor and resolved with time or with conservative management. Practitioner awareness of potential complications will facilitate TTOT candidate selection and the anticipation of postprocedure care problems. (Respir Care 1990;35:153-160.)

Introduction

The demonstrated benefits of treating severe hypoxemia with continuous oxygen supplementation have led to widespread use of home oxygen,[1,2] but difficulties with expense, supply, and patient acceptance have led investigators to seek improved modes of oxygen delivery to ambulatory patients.[3,4] Among these recent innovations, transtracheal oxygen therapy (TTOT) involves the percutaneous insertion into the trachea of a small-diameter, flexible catheter through which oxygen is directly administered. The initial Cleveland Clinic experience with these techniques is reviewed here. Because the advantages of TTOT—improved efficiency of oxygen delivery, enhanced patient compliance, improved mobility outside the home, and improved general functional status and appearance—have been reported,[4] we devote our attention primarily to the adverse events encountered during this experience.

Methods and Materials

Between October 1986 and June 1988, 22 patients who were followed in the Department of Pulmonary Disease at The Cleveland Clinic Foundation underwent 29 oxygen delivery procedures at our institution. Twenty patients had TTOT only, one patient was switched from TTOT to a Johnson Intratracheal Oxygen Catheter (Cook Critical Care,

Dr Adamo is a Fellow; Drs Mehta, Meeker, and Stoller are Staff Physicians; and Ms Stelmach is a Nurse and Respiratory Therapist—Department of Pulmonary Disease, Cleveland Clinic Foundation; Dr Rice is a Staff Physician, Department of Thoracic Surgery—Cleveland Clinic Foundation, Cleveland, Ohio.

The content of this paper was presented in part at the American College of Chest Physicians' 53rd Annual Scientific Assembly, October 26-30, 1987, in Atlanta, Georgia.

Reprints: James K Stoller MD, Cleveland Clinic Foundation, A-90, One Clinic Center, Cleveland OH 44195-5038.

Abbreviations Used in this Paper

P_{aO_2} - Arterial oxygen tension
S_{pO_2} - Oxygen saturation as measured via pulse oximetry
TTOT - Transtracheal oxygen therapy

Bloomington IN), and one patient received only the intratracheal oxygen device. We report only those data that apply to the TTOT. All patients had formerly utilized oxygen via nasal cannulae (mean duration 22.5 months), and all but one were considered stable in their need for oxygen after at least several weeks of medical therapy. One patient's oxygen need steadily rose in the weeks prior to the initiation of TTOT as his disease process (idiopathic pulmonary fibrosis) progressed.

Patients were selected to receive TTOT after meeting all of the following criteria: (1) ongoing need for continuous supplemental oxygen therapy (resting $P_{aO_2} < 55$ torr or pulse oximetry saturation [S_{pO_2}] $< 85\%$ with exercise), (2) previous use of nasal cannulae with inadequate oxygenation on high-flow (≥ 5 L/min) or unacceptable morbidity associated with nasal cannulae therapy (eg, ear and nose soreness, hoarseness, decreased sense of taste and smell, or nighttime displacement from the nares), (3) a high degree of motivation and professed acceptance of the transtracheal system, and (4) no bleeding diathesis or unfavorable neck anatomy. There were 8 women and 13 men, ages 33-74 y (mean 62.2 y), with chronic pulmonary disease (emphysema [13], idiopathic pulmonary fibrosis [3], pulmonary veno-occlusive disease [1], berylliosis [1], primary pulmonary hypertension [1], lymphangioleiomyomatosis [1], and liver disease with intrapulmonary arteriovenous shunt [1]). Twenty patients had a resting $P_{aO_2} < 55$ torr (mean 44 torr), and one patient had an exercise S_{pO_2} of 84% by oximetry. The mean FEV_1 was 1.25 L (range 0.44-3.22 L).

Each TTOT device was placed by a team consisting of a pulmonary physician and a nurse-respiratory therapist. The nurse-respiratory therapist (KS) who assisted in all TTOT-catheter placements in this series had attended a central TTOT training course (Institute for Transtracheal Oxygen Therapy, Denver, Colorado). Of the four physician operators, one physician (ACM) performed most of the TTOT procedures (21/27) and had attended the same central training course.

In addition to hands-on instruction by the centrally trained nurse-therapist and physician, the other three physician operators had reviewed the available training manual[5] and videotape from the vendors of the SCOOP catheter system prior to performing any procedures (Transtracheal Systems, Denver CO). The centrally trained physician directly supervised catheter placement by the other three physicians. Twenty-one patients underwent TTOT catheter placement following the exact published protocol for the insertion of SCOOP catheters.[5] A pre-SCOOP catheter was placed initially, utilizing the needle-guidewire-dilator-catheter system. One week later, an indwelling SCOOP-1 catheter was placed and used for oxygen delivery with the dose of oxygen titrated to maintain an $S_{pO_2} > 90\%$ (Nellcor N-100, Hayward CA). After allowing no less than 6 weeks for the tract to mature, the SCOOP-2 catheter was placed. In addition to assisting in all patient procedures, the nurse-respiratory therapist trained all patients in the care of their catheters, and followed all patients over the duration of the study period. Patient instruction was achieved with printed material, clinic visits, and frequent telephone follow-up. Five patients had a total of six repeat procedures. One patient underwent surgical placement of a Johnson Intratracheal Oxygen Catheter following dislodgment of the SCOOP after 9 months of use. The device and method of implantation have been previously described.[6]

All charts were reviewed for arterial blood gas, oximetry, pulmonary function, and exercise oxygen-titration data. Complications were defined as any development identified by the patient as a problem. Reports of complications were elicited through general inquiries about comfort and catheter caretaking and a specific 'review of systems' inquiry about problems, administered by the same nurse-respiratory therapist who had followed the patients longitudinally. In addition to the complications recorded by attending physicians and the nurse-therapist from direct discussions with patients, charts were reviewed retrospectively (JA) for adverse events.

Results

Table 1 presents the baseline clinical features of the patients studied, including results of measurement

Table 1. Baseline Clinical Features of Patients in the Study Group (n = 21)

	Type of Lung Disease							
	Emphysema n = 13		IPF* n = 3		PVOD n = 1	B n = 1	Lymph n = 1	LD n = 1	PHT n = 1
FEV_1 (L)†	0.86	(0.48-2.11)	1.54	(1.11-1.88)	2.36	1.05	0.88	3.22	2.35
FVC (L)	2.43	(1.15-3.69)	1.77	(1.20-2.33)	3.65	1.28	1.22	4.16	3.35
FEV_1/FVC	0.35	(0.17-0.50)	0.88	(0.81-0.93)	0.65	0.82	0.72	0.77	0.70
Room-Air ABG									
P_{aO_2} (torr)	46	(33-61)	36	(34-38)	51	35	45	41	54
P_{aCO_2} (torr)	42	(27-56)	36	(27-41)	24	47	34	26	35
pH	7.44	(7.37-7.54)	7.45	(7.42-7.49)	7.49	7.40	7.49	7.46	7.44
O_2 Supplement (L/min)‡			6	(n = 2)					
	3	(2-6)	80%	(n = 1)§	3	5	3	6	3

*IPF = idiopathic pulmonary fibrosis.
PVOD = pulmonary veno-occlusive disease.
PHT = primary pulmonary hypertension.
LD = liver disease, with associated arteriovenous shunt.
B = berylliosis.
Lymph = lymphangioleiomyomatosis.
ABG = arterial blood gas values.
†PFT and ABG values are mean (range).
‡To maintain $P_{aO_2} > 55$ torr and/or $S_{pO_2} > 90\%$
§1 of 3 patients with IPF received 80% O_2 by mask.

of airflow and gas exchange, and supplemental oxygen need.

For 20 patients, the mean nasal cannulae oxygen flowrate prior to undergoing TTOT was 3.5 L/min (range 2-6 L/min); one patient required an inspired oxygen concentration of 0.6-0.8 by face mask to achieve an S_{pO_2} of 90%. Mean TTOT flowrate in these 20 patients was 2.2 L/min, which represents a mean reduction in oxygen flow requirement of 37% to provide a similar level of oxygen saturation. Patients had utilized TTOT for a mean of 7.7 months per patient and a total of 162.5 patient-months at the time of this writing. Eleven patients remain on TTOT. Eight patients died (mean 9.5 months following TTOT placement), seven from progression of their underlying lung disease and one from postoperative complications of a bullectomy.

Four patients discontinued TTOT: one patient in the terminal stages of his lung disease after 17 months' use, one patient after the SCOOP-1 catheter became dislodged and the stoma closed, and one patient after repeated dislodgment of the catheter. A fourth patient chose Intratracheal Oxygen Therapy after 9 months of TTOT use because of unwillingness to further care for the SCOOP-2 catheter system. All other patients had been pleased with the TTOT delivery system.

A total of 120 complications were recorded in 21 patients over the 20-month period (162.5 patient-months). This represents an average of 5.7 complications per patient or 0.7 complications per patient-month. Overall, 25 types of complications were encountered and were classified into 4 broad categories (Table 2): Procedural-Technical, Symptomatic, Infectious, and Miscellaneous Complications. For the sake of clarity, we have categorized each adverse event (eg, catheter dislodgment and inability to replace a dislodged catheter are considered separately), avoiding undue redundancy.

Procedural or Technical Complications

The transtracheal catheter was misplaced in two patients, one (SCOOP-2) into the anterior mediastinum (Fig. 1), and one (pre-SCOOP) into the middle mediastinum beside the trachea (Fig. 2). Misplacement of the SCOOP-2 catheter occurred when the patient had difficulty replacing the catheter in a 'mature' tract through which catheters had been replaced twice daily for 4 weeks prior to the incident.

Table 2. Complications of Transtracheal Oxygen Therapy

Type of Complication	Number of Patients (%)
Procedural-Technical	
Misplaced catheter	
Anterior mediastinum	1 (5%)
Middle mediastinum	1 (5%)
Hospitalization secondary to procedural problems (total, 3 days)	3 (14%)
Broken chain	1 (5%)
Dislodged catheter (total, 9 times in 7 patients)	7 (33%)
Repeat procedures (total, 6 procedures in 5 patients)	5 (24%)
Oxygen tubing disconnects from belt clip	8 (38%)
Symptomatic	
Cough	12 (57%)
Excessive sputum	11 (52%)
Transient hoarseness	3 (14%)
Hemoptysis (1 episode required intervention)	5 (23%)
Inspissated mucus plugs	3 (14%)
Subcutaneous emphysema	1 (5%)
Acute respiratory failure	1 (5%)
Excessive stomal granulation tissue	2 (10%)
Intolerance to high-flow TTOT	1 (5%)
Bronchospasm or laryngospasm	2 (10%)
Infectious	
Stomal/neck (total, 8 infections in 6 patients)	6 (29%)
Chondritis	2 (10%)
Additional courses of antibiotics for tracheobronchitis (multiple courses, some patients on cyclic antibiotic schedules)	9 (43%)
Miscellaneous	
Inability to reinsert catheter (total, 11 times in 8 patients)	8 (38%)
Poor catheter care	8 (38%)
Emergency room visits (total, 3 times)	3 (14%)
Unrealized expectations (see text)	3 (14%)
Pneumomediastinum	2 (10%)

This patient was on steroid therapy continuously for chronic obstructive lung disease. Both patients were admitted to the hospital for one day of observation following removal of the misplaced catheter. Neither patient experienced any sequelae following the episodes.

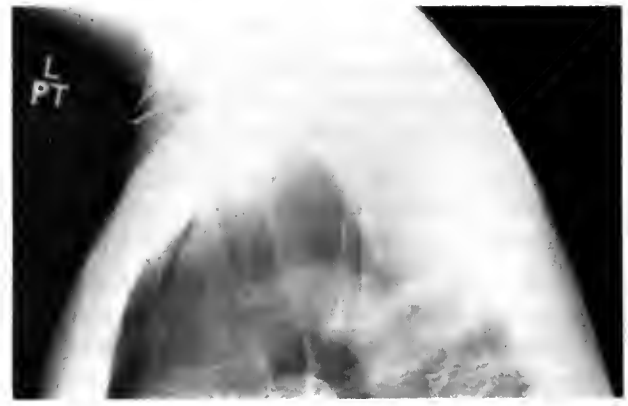

Fig. 1. Lateral chest radiograph showing a misplaced (SCOOP-2) transtracheal catheter in the anterior mediastinum.

Dislodgment of catheters from the stoma occurred nine times in seven patients, usually at night. One dislodgment was the result of a broken neck-chain. Five of these episodes resulted in repeat procedures, two patients refused another procedure and resumed wearing nasal cannulae, and two patients had catheters replaced in the existing tract, with help from a physician.

The other observations in this category were single events.

Fig. 2. Posteroanterior and lateral chest radiograph demonstrating a misplaced transtracheal catheter (pre-SCOOP) in the middle mediastinum.

Symptomatic Complications

Troublesome cough (12 patients) and excessive sputum production (11 patients) were the most frequent problems reported. In all cases, cough and sputum production were deemed to exceed preprocedure frequency and/or severity. Hemoptysis (5 patients) was usually streaky, but was considered substantial (125 mL in one day) in 1 patient and prompted a bronchoscopic examination that revealed friable granulation tissue at the stoma site. Inspissated mucus plugs were seen in 3 patients, and a large inspissated tracheal cast resulted in upper-airway obstruction and acute respiratory failure in 1 patient (the patient developed sudden, extreme respiratory distress with stridor). Urgent bedside bronchoscopy removed this large plug, and the patient's dyspnea, hypoxemia, and hypercapnia readily returned to baseline. It should be noted that this complication occurred in a patient with an oxygen flowrate of 6 L/min who had done well on TTOT for 17 months prior to this incident. Development of the large mucus plug was not related to a known lapse in catheter-cleaning technique or humidification. External stomal granulation tissue in 2 patients was managed with silver nitrate cautery. Intolerance to the sensation of $>$ 1.5 L/min flow of oxygen by TTOT in 1 patient resulted in supplemental nasal cannulae use by the patient.

Infectious Complications

Criteria for defining infection were (1) resolution of warmth and/or erythema (within 2 weeks) in response to additional antibiotics or (2) the presence of gross pus.

Although all TTOT patients received a 7-day course of prophylactic oral antibiotics (cefaclor or cephalexin) following the insertion of the pre-SCOOP catheter, 8 stomal infections were observed in 6 patients. No surgical drainage was required for the 8 stomal infections, all of which resolved within 14 days on oral antibiotic therapy. Chondritis manifested by prolonged tracheal pain at the catheter entry site occurred in 2 patients. At least 9 patients required more frequent use of antibiotics following TTOT compared to their preprocedure baseline, and 2 patients were placed on a cyclic antibiotic schedule (ie, 1 wk/mo) because of repeated episodes of tracheobronchitis with excessive sputum production.

Miscellaneous Complications

Despite careful screening and preprocedure patient education, three patients complained of unrealized expectations. Two were discouraged by catheter dislodgment and refused further TTOT therapy. The third patient later revealed her expectation that TTOT was going to help heal her pulmonary disease rather than merely correct hypoxemia and its attendant morbidity.

Three emergency room visits were attributed to TTOT-associated problems: one visit for pain in the trachea, one visit for accidental dislodgment, and one visit for inability to reinsert the SCOOP-2 during a routine catheter change. One emergency room visit resulted in hospitalization for a total of 2 days due to tracheal pain.

Inability to reinsert the catheter into a developed tract during routine catheter changes occurred 11 times in 8 patients. Only one of these episodes required repeat placement of a pre-SCOOP catheter. All other catheters were eventually replaced in the existing tract, with help from a physician, nurse, or family member. One patient fashioned his own 'guidewire' from a sterilized length of monofilament and rethreaded his own catheter.

Only two anatomic impediments to reinsertion were identified: One patient had excessive stomal granulation tissue (keloid), and another patient developed deviation of the soft tissue tract over time, following considerable weight loss.

Eight patients were unwilling or unable to change or care for the SCOOP-2 catheters on their own.

Of the 120 reported complications, only 10 incidents were considered severe: catheter misplacement, dislodgment with inability to replace without help from medical personnel, significant hemoptysis, and acute hypercapnia. No patient died as a complication of transtracheal oxygen therapy.

Discussion

The use of long-term oxygen therapy has become standard practice in managing severe hypoxemia from chronic pulmonary disease.[3] Continuous administration of oxygen in such circumstances has proven beneficial in terms of improved survival,[1,2] improved exercise tolerance, enhanced neuropsychologic function, reduced erythrocytosis, decreased pulmo-

nary hypertension, and reduced hospitalization.[7-10] The Nocturnal Oxygen Therapy Trial[1] demonstrated the superiority of continuous administration over nocturnal oxygen supplementation alone. The desirability of continuous therapy underscores the importance of patient acceptance and compliance in order to realize the full benefits of oxygen therapy.

Until recently, oxygen administration through nasal cannulae had been the mainstay of home oxygen therapy because of the simple design and ease of use. However, technical and aesthetic considerations detract from the appeal of nasal cannulae. First, oxygen flowrates exceeding 6 L/min through nasal cannulae may be uncomfortable, and the delivered oxygen concentration may fall below needed levels when high flowrates are supplied by an oxygen concentrator. Additionally, a substantial amount of oxygen is wasted into ambient air, particularly during exhalation and swallowing. The high cost of oxygen, averaging approximately $500.00 per month per patient in the study area (2 L/min flow on a liquid oxygen system, 24 hours/day), and the inconvenience of bulky oxygen reservoirs have made oxygen conservation a pressing concern.

Many patients find nasal cannulae (which are draped over the ears, across the face, and under the chin) cosmetically unacceptable. It has been pointed out that cannulae may interfere with the activities of daily living such as showering, shaving, and application of makeup; and they may cause a variety of undesirable events, including ear and nose soreness, hoarseness, and decreased sense of taste and smell, and their use may result in a nighttime displacement of nasal O_2 flow.

In addressing these issues of convenience, comfort, appearance, oxygen conservation, and compliance, several innovations in oxygen delivery have recently emerged—reservoir cannulae,[11,12] inspiratory pulse-flow delivery systems,[13,14] and transtracheal and intratracheal oxygen therapy catheters. TTOT involves the percutaneous insertion of a small-diameter (5-8 Fr) flexible catheter into the trachea through which oxygen is administered. This technique, first described by Heimlich in 1982,[15] can now be performed in an outpatient setting. The SCOOP system was the only TTOT device commercially available in 1989. (Intratracheal Oxygen Therapy, which utilizes a more permanent, surgically placed, subcutaneous catheter, also delivers oxygen directly into the trachea.) Reported advantages of TTOT have included reduction of requisite oxygen flowrates by approximately 50% (allowing adequate oxygenation for those whose needs could not be met by high flowrates with nasal cannulae), greater mobility outside the home (portable oxygen supplies last longer with reduced oxygen flowrates), improved appearance (the TTOT catheter is readily hidden beneath a scarf or collar and the oxygen tubing beneath clothing), improved compliance with oxygen prescription, improved exercise tolerance, and an enhanced self-image.[4,16-20]

Offsetting these widely accepted advantages of TTOT to some extent are the associated adverse effects that have also been described since the first reports of its use. These include complications related to procedural misadventures, equipment breakage, dislodgment of catheters, infection and irritation, and care and cleaning difficulties.[21-24] Previously reported complications of TTOT that were not encountered in our patient series include cephalad displacement of the catheter, catheter breakage, an increased frequency of respiratory tract infections requiring hospitalization and intravenous antibiotics,[25] candidal stomal dermatitis and cellulitis, contact stomal dermatitis, cutaneous bleeding, and cardiac arrhythmias.

The goal of the current research was to assess comprehensively adverse experiences associated with the TTOT system during the start-up experience at The Cleveland Clinic Foundation, a large tertiary care facility. Our findings support prior observations that although some adverse events are usual, clinically serious complications are uncommon (10/120 complications in the initial experience reported here). Furthermore, the occurrence of minor complications does not discourage most patients from continuing use of transtracheal oxygen. Only 3 of the 21 patients initiating transtracheal oxygen therapy in the current series elected to discontinue its use in favor of nasal cannulae; one patient elected to switch to intratracheal oxygen therapy.

Several more substantial complications were observed in our series and deserve further comment. The operator was responsible in only one of the two catheter misplacements into the mediastinum. In the second instance, a patient's attempt to replace the catheter into a putatively mature tract resulted in

extratracheal dissection into the pretracheal space. This experience underscores the importance of visualizing the stoma as clearly as possible and of avoiding the exertion of more than usual force in catheter replacement.

The development of a large tracheal cast in one patient in this series recalls a previously cited experience.[26] In our patient, the tracheal cast developed in an experienced patient after longstanding catheter use (17 months), despite the use of humidification and catheter care that was considered appropriate. However, the requisite oxygen flowrate through the transtracheal oxygen catheter was 6 L/min. In our opinion, this event justifies increased clinical vigilance in those patients whose oxygen flowrate through the transtracheal catheter is high (ie, $>$ 3 L/min).

Despite our reporting on only 21 patients in this series, the following considerations suggest that this experience can be generalized to other large centers that are also undertaking a TTOT program:

1. All catheters were placed under the direct supervision of a trained physician and nurse-therapist, both of whom had carefully studied the available manuals and had participated in central training programs.
2. Although our patients were sick (as manifested by the high mortality rate), they appear to be representative of patients in tertiary centers whose selection for transtracheal oxygen therapy is based on stringent criteria for improved oxygen delivery rather than cosmetic considerations alone.

In our series, all catheters were placed under the direct supervision of centrally trained operators. To the extent that central training of operators is essential to optimal catheter placement technique, it is possible that operators with less training may experience more complications in their early experience than were observed in our series. On the other hand, if TTOT is to achieve the same widespread clinical use as other invasive pulmonary techniques widely used in practice (eg, bronchoscopy and pleural biopsy), it seems unreasonable to expect that all operators will receive central training, but rather that appropriate placement technique will be disseminated by review of complete published materials and hands-on instruction by more-experienced operators.

Several cautions are noteworthy in interpreting our study results. First, because we report on the first 21 patients to be offered TTOT at the Cleveland Clinic, these observations most aptly reflect a start-up experience. While it is tempting to examine this experience for evidence of a 'learning curve,' no such trend is apparent here, and the number of study participants seems to be too small to permit meaningful subgroup analyses. The current study was not designed to formally analyze the cost effectiveness and risk-benefit tradeoffs of TTOT vs oxygen delivery by nasal cannulae, but such analyses are needed before such newer technologies can be meaningfully endorsed.

In summary, this initial experience with 21 patients at the Cleveland Clinic Foundation confirms other previous observations that oxygen administration with the SCOOP transtracheal catheter is a useful strategy for providing supplemental oxygen when this need cannot be satisfactorily met with traditional methods (eg, nasal cannulae). Although frequent, the minor adverse events that were observed did not discourage continued use of transtracheal oxygen in most patients. The possibility of more serious complications has encouraged us to continue to monitor adverse events with special attention to whether adverse events observed in this study will become less frequent with increased experience. Although we believe that the experience reported here can be generalized to other centers starting TTOT programs (based on the representative nature of the patients and the training experience of the operators in this study), we suggest that before the technique can be widely embraced, similar monitoring of patients' experiences with TTOT should be undertaken.

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The Prediction and Prevention of ARDS

Leonard D Hudson MD

Introduction

The death rate of patients with the adult respiratory distress syndrome (ARDS) continues to be high—in excess of 60%. This high mortality is not due to failure of our current means of respiratory care and respiratory support. Only a small percentage, approximately 10%, of patients dying with the adult respiratory distress syndrome die a true respiratory death—that is, one due to refractory hypoxemia or respiratory acidosis.[1] The mortality associated with ARDS has not changed substantially since the syndrome was recognized and the term coined in 1967.[2] Mortality has not changed despite the fact that respiratory care has become much more sophisticated during this time period, and many advances have been made in our ability to support a failing respiratory system. In fact, I believe that one of the reasons that the mortality rate has not changed is that we now have the ability to support acutely ill and injured patients—because of improvements in respiratory care and other medical and surgical advances—so that now these patients survive long enough to develop ARDS when previously they would have died before it could be detected. It is my contention that patients developing ARDS now are apt to be more severely ill, on the average, than were patients developing ARDS 15 to 20 years ago. Thus, although it is theoretically possible that improved respiratory support could reduce mortality in ARDS patients—as I will discuss—the impact of such improved support is likely to be relatively small because patients usually die of causes other than refractory hypoxemia or respiratory acidosis.

We are left then with a dilemma—how do we reduce the mortality associated with ARDS? The major strategy proposed to address this dilemma has been institution of early therapeutic interventions that would either prevent the development of ARDS or modify its severity. Such prevention or early intervention has two major prerequisites: the ability to identify patients who are at high risk for developing ARDS and the availability of safe and effective interventions. This strategy to prevent or ameliorate ARDS was first proposed many years ago. Over the last decade much research has been aimed at achieving these two prerequisites. I will review the current state in terms of the prediction and prevention of the adult respiratory distress syndrome.

Prediction of ARDS

Rationale

Several rational reasons exist for wanting to be able to predict which patients are at high risk for developing ARDS. This knowledge would help us understand the epidemiology of ARDS and its pathogenesis. Clinical management could be improved if we had this knowledge; we would be better able to discuss prognosis with the patient and family, and high-risk patients could be identified for more intensive monitoring and supportive therapeutic measures. Finally, and perhaps most importantly, prediction of patients at high risk would allow specific early therapeutic interventions to prevent or modify the development of this syndrome. As of 1989, no such beneficial intervention had been proven. However, information on the patient at high risk for developing ARDS is crucial in order to be able to

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A version of this paper was presented as the Egan Lecture at the Annual Meeting of the American Association for Respiratory Care on December 3, 1989, in Anaheim, California.

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Abbreviations Used in this Paper

anti-TNF	- Anti-tumor necrosis factor
ARDS	- Adult respiratory distress syndrome
BAL	- Bronchoalveolar lavage
CFI	- Chemotactic factor inhibitor
CPAP	- Continuous positive airway pressure
ECMO	- Extracorporeal membrane oxygenation
$ECCO_2R$	- Extracorporeal CO_2 removal
IRV	- Inverse-ratio ventilation
NSAIDs	- Nonsteroidal anti-inflammatory drugs
PAF	- Platelet-activating factor
P_{aO_2}	- Arterial oxygen tension
PEEP	- Positive end-expiratory pressure
PET	- Positive emission tomography
SDD	- Selective decontamination of the digestive tract

carry out appropriate clinical trials of several proposed interventions.

Knowledge of the true risk for development of ARDS is important in carrying out clinical trials, for several reasons. First, a true benefit may be missed if an inappropriate study population in which ARDS incidence is extremely low is used. The use of an inappropriate study population would make it more difficult to recognize even a substantial benefit, and the study population size would have to be very large, making the clinical trial much more difficult to carry out. Second, early intervention may be necessary in order to have any effect on modifying the syndrome. (Once lung injury is well established, it may be much more difficult to show a positive therapeutic effect.) And, finally, a therapeutic trial in patients not at risk of developing ARDS would be inappropriate and even unethical; if the study medication or intervention carries any risk, treating a patient with no chance of developing the condition is unwarranted.

Three approaches to prediction or early detection have been attempted: (1) measurement of physiologic changes or abnormalities; (2) measurement of mediators or markers of ARDS present in the blood of patients at high risk; and (3) identification of clinical predispositions or risk factors for the development of ARDS. Of these three approaches, the identification of clinical predisposition is the one that has provided the most useful information for carrying out clinical intervention trials. Each of these approaches will be discussed and examples of their applications given.

Measurement of Physiologic Derangements in the Prediction of ARDS

In general, this approach has looked for physiologic abnormalities in patients who go on to develop ARDS compared to those who do not. Although the mean value for a given measurement may be statistically different between the two groups, considerable overlap usually exists. Such overlap makes it difficult to apply these data to an individual patient. Another significant limitation of this approach is that 'prediction' by physiologic derangement probably represents early detection of patients with lung injury. The physiologic variable often reflects that a degree of acute lung injury is already present. It would be ideal if one could identify the patient at high risk before substantial lung injury has occurred.

Shoemaker and associates investigated 152 patients with multiple trauma and shock.[3] Of these, 39% subsequently developed acute respiratory failure. Shoemaker et al found no differences in hemodynamic measurements or in pulmonary gas exchange between those who did and those who did not develop respiratory failure. They did find that, on the average, patients who developed respiratory failure had (1) a lower blood volume and red-cell mass and (2) lower oxygen delivery. Blood volume and red-cell mass are difficult measurements to make and do not lend themselves to convenient screening for ARDS risk. Even if the measurements were more clinically applicable, overlap in values of these measurements was present between the two groups (ie, values on the low end of the range for those who did not develop failure were not different from values on the high end of the range for those who did). The finding of lower oxygen delivery in patients who developed ARDS is of interest because the two components determining oxygen delivery (cardiac output and arterial oxygen content) were not significantly different in the two groups. Again substantial overlap severely limits application of this finding for prediction of ARDS in the individual patient.

Weigelt and his associates[4] studied trauma and surgical patients at considerable risk for ARDS development. Differences in intrapulmonary shunt and total static compliance existed between those who went on to develop ARDS and those who did not (Fig. 1). Again, although the mean values differed, overlap severely limits the application of this

information in a specific patient in terms of predicting the likelihood of ARDS.

Because of these limitations, it is unlikely that using physiologic variables to predict development of ARDS in the individual patient will be fruitful. This does not preclude the use of sophisticated physiologic imaging approaches to early detection of lung injury and edema. Three general approaches have been attempted: (1) measurement of the clearance of radiolabeled aerosols by the lungs; (2) measurement of the leakage of labeled macromolecules from the pulmonary microcirculation; and (3) imaging techniques to detect excess lung water and labeled solutes in the lung. One such approach is the use of positive emission tomography (PET) to detect the pulmonary transcapillary leakage rate in patients at risk of developing ARDS.[5] Although Schuster and his colleagues at Barnes Hospital and Washington University School of Medicine in St Louis have been successful in early identification of an increased pulmonary transcapillary leakage rate, this approach is limited at this time by its expense and lack of availability. In 1989, PET scans were available only in major research institutions. Even in those institutions, critically ill patients have to be transported to the imaging facility to have these measurements made. At Barnes Hospital, a PET unit is dedicated to the intensive care unit and located adjacent to it, but I know of no other similar situation. We all hope that one of these imaging approaches will prove to be a practical means of identifying developing lung injury at an early stage. Measurement of excess lung water has not achieved this goal because the range of normal values for lung water is large, and thus the results of a single measurement in an individual patient may have little meaning.

Measurement of Blood-Borne Mediators or Markers of Acute Lung Injury

It is generally thought that ARDS results from a systemic intravascular inflammatory process that escapes its usual controls and leads to multisystem organ injury.[6–8] The lungs often sustain the most severe injury. In fact, frequently the lungs may be the only organ system that has clinically apparent injury as manifested by the development of organ failure

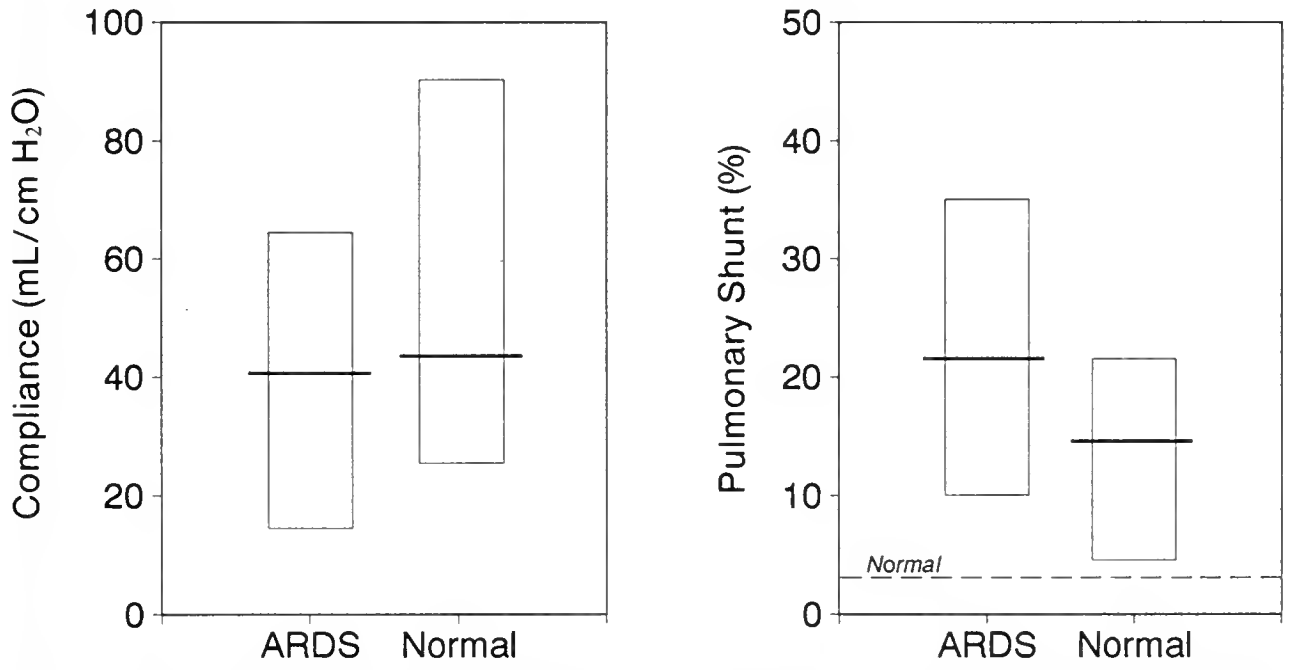

Fig. 1. (A) Values for total respiratory compliance (mL/cm H_2O) and (B) pulmonary shunt (%) for patients with severe trauma, abdominal sepsis, and/or aspiration of gastric contents, with patients stratified according to whether they subsequently developed criteria for ARDS.[4] Overlap is present for both physiologic measurements between the two patient groups, and the mean values for compliance are similar.

(ARDS).[9] This intravascular inflammatory process involves an increase or activation (or in a few cases a decrease) in a variety of mediators or potential markers of the inflammatory process, and subsequent lung injury. Some of these potential mediators or markers of acute lung injury are shown in Table 1.

Table 1. Blood-Borne Mediators or Markers of Acute Lung Injury

Formed blood elements
Neutrophils
Platelets
Chemoattractants
Complement fragments
Lipoxygenase products
Platelet-activating factor
Bacterial peptides
Mediators of cell damage
Toxic oxygen intermediates
Proteolytic enzymes
Phospholipase products
Tumor necrosis factor
Markers of cell damage
Prostacyclin
Factor VIII

They include formed blood elements that can participate in the process, particularly neutrophils.[10-13] Neutrophils in ARDS may become sticky and adhere to the endothelial surface of small vessels in the lung, releasing injurious substances. Studies have attempted to measure differences between the number of neutrophils entering and leaving the lung, with the difference presumably reflecting an increased adherence of neutrophils in the lung microvasculature.[14]

Several chemicals that have chemoattractive (ie, chemotactic) properties can be involved. These substances frequently are early players in the process attracting neutrophils or platelets to the area of injury and activating the potential of these cells to release toxic substances. In addition to naturally occurring chemicals that act as chemoattractants, sepsis due to certain microorganisms results in the release of bacterial peptides that also act as chemoattractants.

Several substances are involved as mediators of the direct cell damage.[6-8,15-17] These include toxic oxygen radicals, proteolytic enzymes, and phospholipase products, all of which can be released by activated neutrophils. Tumor necrosis factor produced by macrophages appears to be an extremely important mediator of cellular injury in sepsis. Prostacyclin and coagulation factor VIII are products that result from cellular damage and thus can serve as markers of this injury. Fibronectin is a naturally occurring substance that combines with bacteria in the bloodstream (so-called opsonization). Opsonization aids the ingestion and destruction of bacteria by neutrophils and macrophages. When fibronectin is reduced in quantity, the risk of sepsis and subsequent lung injury is enhanced.

To date, measurement of blood-borne mediators or markers of acute lung injury has not been useful in predicting which patients will develop ARDS. Several factors have been found to be abnormal in patients with ARDS. However, studies using appropriate control populations (ie, patients with similar illnesses and injuries) have found that high-risk patients who do not subsequently develop ARDS also have a significant increase or activation of the measured blood constituent. An example of this is the story of the measurement of activated complement. A study in 1980 by Hammerschmidt and colleagues,[18] in which our ARDS investigation team participated, provided the exciting finding that activated complement levels were elevated in patients developing ARDS but were normal in other critically ill patients in an intensive care unit. At that time, we were limited in our knowledge of clinical predispositions that placed patients at high risk for ARDS. In subsequent work with appropriate controls, both our group and others have found that activated complement is found in patients with clinical predispositions who do not go on to develop ARDS as well as in those who do.[19-21] Thus, although activation of complement may be a very important step in the systemic inflammatory process and may participate in lung injury, its identification is insufficient to allow prediction that an individual patient will go on to develop ARDS.

The measurement of factor VIII, a marker of endothelial cell injury, may prove to be an exception to the heretofore futile attempts to identify markers that predict development of ARDS. Workers at the University of California at San Francisco have found in a preliminary study of patients with sepsis syndrome

that those who have progression in lung injury (as determined by a composite injury score) have significantly elevated factor-VIII antigen levels compared to those without progression in the lung injury score.[22] To date these results have been presented only in abstract form, with a limited number of patients, but further work is ongoing. Because factor-VIII antigen reflects endothelial injury, it is possible that this is a relatively late step in the lung injury process; its presence may be less useful for predicting the development of ARDS than as a marker of lung injury before full-blown ARDS is actually present. Also, at the present time, the laboratory measurement of factor-VIII antigen involves an incubation step that requires 12 to 18 hours. This time period limits the utility of the test as a predictive tool, although, presumably, a more rapid test could be developed.

Most of the studies of blood-borne substances as predictors of ARDS development have looked at pro-inflammatory factors. The possibility exists that anti-inflammatory factors may be reduced, and, thus, the balance between pro- and anti-inflammatory factors is shifted toward pro-inflammatory activity. A study by workers at the University of Washington and University of Nebraska, also reported only in abstract form, has evaluated the presence of chemotactic factor inhibitor (CFI) in the blood of patients at risk for ARDS.[23] Their findings suggest that, although CFI levels are markedly increased in the bronchoalveolar lavage (BAL) fluid of patients who go on to develop ARDS, the actual functional activity of CFI in this fluid is reduced. This finding is impractical for prediction of ARDS because the measurement requires BAL fluid rather than blood. However, this finding supports the notion that it may be important to evaluate anti-inflammatory factors in looking at the overall inflammatory-state balance.

Clinical Predispositions in the Prediction of ARDS

Until recently, most of the clinical predispositions to ARDS were determined by retrospective analysis of patients who developed ARDS.[24] These studies did not allow determination of the true incidence of ARDS associated with any of the clinical predispositions. The first prospective studies looking at association of ARDS development with clinical predisposition were performed in the early 1980s. Our investigative group at the University of Washington, in a study by Pepe et al,[25] used a relatively exclusive approach in an attempt to identify a high-risk population of patients in preparation for a clinical intervention trial. By exclusive I mean that there were several exclusion criteria, and only those patients with factors that placed them at a fairly high risk for developing ARDS were included. With this approach, we identified a relatively high incidence of patients who went on to develop ARDS (34%); however, this approach lacked sensitivity in that these clinical risks and exclusions failed to identify over half of the patients who went on to develop ARDS. At about this same time, a study by investigators at the University of Colorado used an inclusive approach in an attempt to identify all of the clinical predispositions that placed patients at risk for developing ARDS.[26] With this approach, they found a relatively low incidence of ARDS (6.8%) but 'missed' relatively few cases (approximately 20%).

Based on the results of these two studies, we set out to use risk-factor definitions with the goal of retaining the ability to identify a relatively high incidence of ARDS and yet to improve our sensitivity, that is to have relatively few missed cases. We initially included eight risk factors—four primarily medical and four primarily surgical. The medical risk factors included sepsis syndrome, aspiration of gastric contents, drug overdose requiring mechanical ventilation, and near-drowning. Of these, sepsis syndrome obviously also occurs in surgical patients. We later deleted near-drowning as a risk, not because of a lack of association with ARDS but simply because we had too few patients with this predisposition to allow a meaningful analysis. The 'surgical' risks included pulmonary contusion, multiple major fractures, multiple transfusions for emergency rescuscitation, and head trauma. After collecting a large number of patients with head trauma, we found that the incidence of ARDS following isolated head trauma was low, and the occurrence of head trauma in addition to any other risks did not add to the likelihood of developing ARDS. Therefore, we omitted patients with isolated head trauma from data analysis in order to achieve a higher overall incidence of ARDS. For all of these predispositions, we had very specific definitions that have been reviewed

elsewhere.[25,27,28] The definition of sepsis syndrome is worth noting because it differed from the related risk factor used in the University of Colorado study.[26] We used a clinical definition of sepsis syndrome or septic shock that could include positive blood cultures or other positive cultures, but did not require the presence of those positive cultures[25,27] (being aware that approximately one half of the patients that we considered to have septic shock on a clinical basis had negative blood cultures for various reasons).[25,28] The study by Fowler and colleagues[26] evaluated the presence of bacteremia, defined as the presence of two positive blood cultures, as a risk factor.

The results of our revised study are illustrated in Figure 2. The highest incidence of ARDS was found in patients with sepsis syndrome—nearly 40%. Surgically related factors had an intermediate incidence of approximately 15 to 20%, whereas aspiration of gastric contents and drug overdose were associated with an incidence of approximately 10%. The simultaneous occurrence of more than one risk factor more than doubled the incidence of ARDS. Therefore, by using a single predisposition of sepsis syndrome or a combination of any of the other predispositions, a 40 to 50% risk of ARDS development could be identified. This incidence is sufficient to allow a valid study design for clinical trials of ARDS intervention. During the same time period, we missed only about 20% of the patients who developed ARDS. Thus, the sensitivity was markedly improved in this study as compared to our previous investigation.[25]

Clinical predispositions allow prediction of a relatively high incidence of ARDS (20 to 50%, depending on the risk factor or combination of risk factors used). It is possible to use a group of definitions that result in missing relatively few patients who go on to develop ARDS. Multiple risks predict a higher incidence.

The advantages of using clinical predispositions as predictors include their objective and reproducible nature, if very specific definitions are employed. Also, no invasive tests are required. The limitations of this approach include the relatively low specificity. Even with the use of a combination of risk factors, approximately one half of the patients identified as being at risk will not develop ARDS. In addition, the use of multiple clinical predispositions may result in selection of a heterogeneous population that can have drawbacks in clinical intervention trials. For example, a particular pharmacologic intervention may be helpful in patients with trauma as a clinical predisposition, but may not prove beneficial in patients with aspiration of gastric contents. Unless this is taken into account in the study design, the beneficial effect of a particular drug in a certain category of patients may be diluted and, therefore, not recognized. As with the other approaches, use of some of the clinical risk factors results in the identification of patients who have already developed some degree of lung injury. This is particularly true of sepsis syndrome as a clinical risk. By the time the criteria for sepsis syndrome are met, the systemic inflammatory response may be far-advanced. Frequently, a patient has already developed a degree of lung injury, and occasionally full-blown ARDS is already present.[29] On the other hand, some of the definitions that identify patients with severe trauma can be recognized early (presumably even at the scene of the accident) and thus allow early clinical intervention. We and our

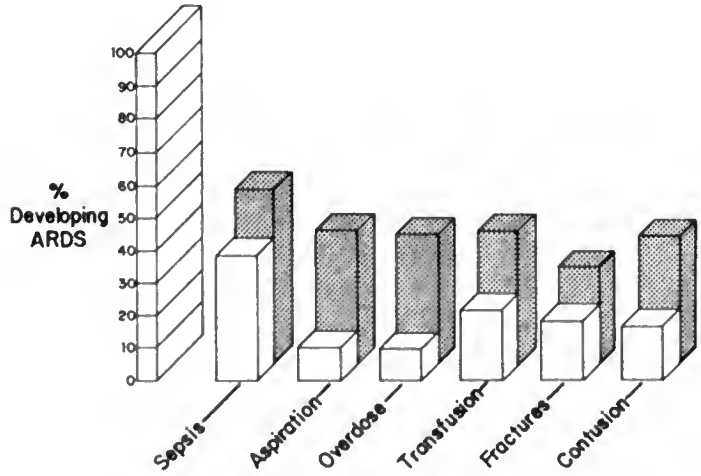

Fig. 2. The incidence of ARDS in patients meeting predefined criteria for various risk factors either alone (open boxes) or in combination with other risk factors (shaded boxes).

fellow investigators currently believe that identification of clinical predispositions is the most practical method of predicting development of ARDS for the purpose of performing randomized clinical trials.

Prevention or Early Treatment of ARDS

It is problematic whether true prevention of acute lung injury can be achieved. The identification of several of the clinical predispositions for ARDS logically suggests that the process of lung injury has already been initiated. This is particularly true for the risks of aspiration of gastric contents and sepsis syndrome. With any of the risks, a degree of lung injury may already be present when the risks are first recognized. Thus, true prevention may be impossible. However modification of the lung injury is certainly a possibility with the institution of early therapy. Early therapy could result in less severe lung injury (so that full-blown ARDS might not develop or would be less severe than if interventional therapy had not been initiated), a shorter course of respiratory failure, the development of fewer complications, and improved survival.

At the present time no clinical intervention has been identified that results in any of these desired goals. However, three strategies in early treatment of ARDS can be identified: (1) correction of physiologic derangements, (2) suppression of alveolar inflammation, and (3) the prevention of complications. I will summarize each of these three approaches and give examples of potential interventions. Several of the pharmacologic approaches are dealt with in greater detail elsewhere.[30]

Correction of Physiologic Derangements

The possible interventions aimed at correcting physiologic derangements include: (1) early application of positive end-expiratory pressure (PEEP); (2) early application of newer ventilatory modes including extracorporeal CO_2 removal ($ECCO_2R$) and inverse-ratio ventilation (IRV); (3) administration of aerosolized surfactant; and (4) administration of almitrine, a drug that augments hypoxic vasoconstriction, which I will not discuss further.

Early Application of PEEP. Our group of ARDS researchers at Harborview Medical Center and the University of Washington in Seattle studied whether the early application of PEEP in patients at high risk for ARDS would prevent the development of the syndrome.[27] Previous studies had suggested that so-called prophylactic PEEP did prevent ARDS by showing improved oxygenation in the PEEP group compared to controls.[31,32] Our question was not whether PEEP would improve oxygenation in those patients developing some degree of lung injury (we assumed that this would be the case), but whether early application of PEEP would modify the tissue injury process so that the pathophysiologic syndrome of ARDS would be prevented or ameliorated. Therefore, in order to study the treatment and control groups under the same conditions, we compared arterial blood gas values in both groups after removing PEEP for a 5-minute period at 24 hours following PEEP application and each 12 hours thereafter. One of the earlier investigations had studied patients who did not have generally accepted risk factors for ARDS, but who did have risks for developing atelectasis, particularly postoperative atelectasis.[31] Their favorable results can be interpreted as possibly preventing atelectasis instead of ARDS. The other major study clearly included surgical and trauma patients at high risk of ARDS development.[32] In this study, the same number of patients developed some degree of lung injury in both the treatment and control groups, but oxygenation was more abnormal in the control group. Because measurements were made on PEEP in the treatment group and without PEEP in the control group, the results may have reflected a higher P_{aO_2} from the PEEP, even if there was no difference in the degree of lung injury between the two groups.

We studied patients shown in a previous investigation to be at high risk for developing ARDS, as described above.[25] We also controlled for mechanical ventilation, placing all subjects in both groups on intermittent mandatory ventilation with at least four mandatory breaths per minute. With this study design, we applied 8-cm-H_2O PEEP (the level which was purported to markedly reduce ARDS incidence in the study by Schmidt et al[31]) and found no difference in the development of ARDS. In the early PEEP group, 25% of patients developed ARDS compared to 27% in the control group. In addition, we failed to find a difference either in the development of complications (including all pulmonary complications) or mortality between the two groups. We

concluded that early application of PEEP does not alter the natural course of acute lung injury when applied to patients with clinical predispositions for the syndrome.[27]

New Modes of Respiratory Support. In recent years, new ventilatory modes have been proposed for the management of ARDS. If these modes are applied early to patients at risk of developing ARDS or with early manifestations of the syndrome, will they reduce mortality associated with ARDS? We have previously demonstrated that true respiratory deaths (defined as dying of refractory hypoxemia or respiratory acidemia despite maximal attempts at oxygenation and ventilation) are relatively unusual and account for only about 10% of all deaths in ARDS.[1] This implies that our current means of respiratory support are relatively successful and suggests that improved ventilatory modes might have relatively little impact on mortality. However, another possible hypothesis exists that is compatible with the possibility that improved ventilatory support could, in fact, reduce mortality. We and others found that the majority of patients dying with ARDS either died of their underlying illness or injury, which presumably could not be prevented by better ARDS management, or died with sepsis syndrome and accompanying multiple organ-system failure.[1,33,34] Further, we surmised that sepsis syndrome occurred in those developing nosocomial pulmonary infections. If the acute lung injury could be lessened in severity or the course of the injury could be shortened, then patients would be less likely to develop these fatal complications and mortality might improve. Thus, if current ventilatory modes compound the lung injury—perhaps by using relatively large tidal volumes when the available lung volume has been decreased by pulmonary edema and atelectasis—any ventilatory means that would avoid worsening the lung injury might result in a less severe and shorter course of ARDS and, thus, reduce the likelihood of developing fatal complications.

Gattinoni and co-workers[35] in Milan, Italy, have suggested that this is the case. They have used a form of respiratory support that is a modification of apneic oxygenation and $ECCO_2R$. A low level of PEEP or CPAP and a low frequency (3 to 4 breaths per minute) of low-volume breaths are applied to prevent progressive atelectasis. CO_2 removal is carried out by a veno-venous bypass method that involves an extracorporeal permeable membrane. They applied this method to patients with severe ARDS who met the criteria of the decade-old multicenter extracorporeal membrane oxygenation (ECMO) study performed in the United States.[36] Gattinoni et al reported a marked reduction in mortality in their patients compared to the historical control group from the ECMO study.[35] This means of respiratory support requires sophisticated personnel and monitoring and should not be widely applied without a study involving a concurrent control group. Such a study is currently being carried out at the University of Utah.[37] Even if this method proves successful, it cannot be considered a preventive technique or even an early intervention method as the Gattinoni group applied it only after a subject's lung condition worsened during more conventional mechanical ventilation.

Another relatively new method of ventilation that has been applied in patients with ARDS is inverse-ratio ventilation (IRV) in which the inhalation phase of ventilation is prolonged, and a pressure control mode of ventilation is utilized.[38] IRV may occasionally improve oxygenation in patients with severe ARDS in whom conventional modes of ventilation are failing; however, we know of no logical reason to suggest that this mode of ventilation would have any substantial benefit over more conventional methods if applied early in patients with developing lung injury.

If new ventilatory methods are to reduce mortality in ARDS, they must result either in a less severe degree of lung injury or shorten the course of ARDS. Although this remains possible, it is not likely that application of any particular ventilatory technique to the patient at risk for developing ARDS will substantially affect outcome.

Surfactant Administration. Surfactant administration or replacement is of considerable interest as a means of improving the physiologic derangements in ARDS. The administration of surfactant to animal models with lung injury has resulted in improved oxygenation and compliance. Successful trials in neonates with infant respiratory distress syndrome have further kindled this enthusiasm.[39,40] Several

questions remain in regard to administration of surfactant to adults with ARDS. First, a question exists as to which product might be most advantageous. Artificial surfactant products with and without a protein moiety are available as are natural surfactants from animal sources. Although the natural surfactants may have some advantage in physiologic effect, their antigenic nature carries potential risks.[41] Second, delivery problems must be addressed. A relatively large volume of the surfactant product must be delivered. Most forms of surfactant are relatively viscous in nature, and the optimum means of delivery has not yet been determined. Third, it is not known whether early administration of surfactant in patients at high risk for developing ARDS would carry any advantage over delivering it to patients who already have evidence of significant lung injury.

Strategies To Prevent or Reduce Alveolar Inflammation in ARDS

Several potential inhibitors of inflammatory lung injury in ARDS have been proposed. These drugs fall into two main groups: (1) nonspecific anti-inflammatory drugs including corticosteroids, nonsteroidal anti-inflammatory drugs (NSAIDs), and pentoxifylline, and (2) direct inhibitors or scavengers of inflammatory agents. This latter group includes antiproteases, antioxidants, platelet-activating factor (PAF) antagonists, and specific antibodies against toxic inflammatory agents including anti-tumor necrosis factor (anti-TNF), anti-endotoxins, and anti-C5a. These agents are discussed in more detail elsewhere.[30]

Steroids have been the most widely studied agents in both treating and preventing ARDS. No benefit has been demonstrated, either in ARDS treatment[42,43] or in ARDS prevention.[29,44] The prevention of ARDS has been studied in two patient populations—patients with sepsis syndrome[29] and patients with multiple trauma or postoperative conditions placing them at high risk for ARDS development.[44] Luce and co-workers[29] found no difference in the rate of ARDS development in patients with sepsis syndrome. They also failed to find differences in mortality and oxygenation status between the treatment and control groups (Fig. 3). Weigelt and colleagues[44] studied surgical and trauma patients at high risk for ARDS. Patients receiving 48 hours of intravenous steroids had a tendency to develop a more severe degree of lung injury, compared to those receiving placebo. Infectious complications at 1 week were significantly increased in the steroid-treated group compared to the control group. A randomized trial of corticosteroids in patients with fractures at risk for fat-embolism syndrome found that corticosteroid administration resulted in a lower incidence of a largely subclinical fat-embolism syndrome.[45] However, this finding was of questionable clinical importance, and no deaths occurred in either group. Thus, there is no compelling evidence that corticosteroids prevent development of ARDS, and one study demonstrated an increased incidence of infectious complications with steroid treatment.[44]

Of the NSAIDs, ibuprofen has been widely studied in animal models with significant beneficial effects. A pilot project has been carried out in human subjects with ARDS in preparation for a multicenter clinical trial on a larger scale. In the pilot phase, both patients with sepsis syndrome at high risk for developing ARDS and patients with ARDS already present were studied. The most striking finding was increased resolution of ARDS. However, there was also a tendency towards fewer at-risk patients developing ARDS in the treatment group. Both of these findings were based on insufficient numbers of patients to allow any firm conclusions, but they do warrant further clinical investigations of both treatment and prevention.

Pentoxifylline has several anti-inflammatory actions. Pretreatment in an experimental model of sepsis has demonstrated protection against develop-

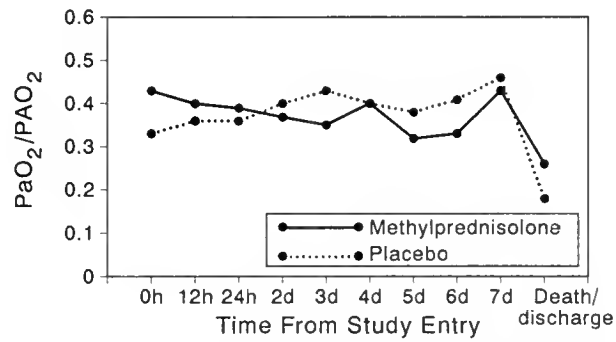

Fig. 3. Oxygenation as determined by P_{aO_2}/P_{AO_2} was similar for patients with clinical sepsis whether they received methylprednisolone or placebo.[42]

ment of lung injury.[46] However, no human studies are available.

Both proteases and oxidants (particularly toxic oxygen radicals) are thought to play important roles in mediating direct tissue injury. Administration of alpha$_1$ protease inhibitor (alpha$_1$-PI) is known to be safe from studies in patients with alpha$_1$-antitrypsin deficiency.[47] Feasibility studies for alpha$_1$-PI administration in patients with ARDS are being carried out. One of the questions regarding efficacy is raised by the observation that alpha$_1$-PI is already abundant in trauma patients. Thus, it is unclear whether a further increase in these amounts will be of benefit. The antioxidants superoxide dismutase and catalase have been shown to prevent lung injury in animal models.[48,49] However, these antioxidants are difficult to deliver to sites where they might be effective and are costly to produce. N-acetylcysteine, a commercially available antioxidant, has been shown to reduce lung injury in a sheep sepsis model[50] and is under study in human ARDS in Europe.

A polyclonal antibody to endotoxin has been shown both to reduce mortality in septic shock in human patients[51] and also to prevent development of shock in high-risk surgical patients.[52] The means are now available to produce large quantities of monoclonal antibodies against specific substances. Two clinical trials using a monoclonal antibody against endotoxin have recently been completed, but the results of those trials are not yet available. A monoclonal antibody against tumor necrosis factor has been shown to reduce shock-related organ failure and mortality in an animal model of sepsis.[53] Other means of counteracting the clinical effects of endotoxin are also available including the use of detoxified endotoxins or endotoxin analogues that compete with bacterial endotoxins for endotoxin receptors and thus reduce the toxic effects of endotoxin.

Strategies To Prevent Complications of ARDS

Infectious complications appear to be the most common and the most severe complications occurring in patients with ARDS.[1,33,34] Infections (in particular nosocomial pneumonias) are associated with septic shock and multiple organ-system failure and a high mortality rate.[1,33] Therefore, the primary strategies for preventing ARDS complications and improving survival should be aimed at preventing infectious complications in patients with ARDS. Two major strategies are being proposed—the administration of sucralfate for the prevention of gastric bleeding and an overall infection-prevention strategy called selective decontamination of the digestive tract, or SDD. Both of these are discussed in more detail elsewhere.[30]

Patients with acute respiratory failure have a high incidence of gastrointestinal (GI) bleeding,[54] although this has lessened in recent years both in incidence and severity. GI bleeding has been successfully prevented with administration of antacids or H-2 blockers, both of which reduce gastric acidity. Studies have produced circumstantial evidence that bacterial colonization of the stomach is associated with more frequent tracheal colonization and development of nosocomial pneumonias[55–57] and also that preservation of gastric acidity prevents gastric colonization.[58,59] Based on these rationales, two studies have compared the administration of sucralfate to other means of preventing GI bleeding. One study compared sucralfate either to antacids or H-2 blockers, and found a strong trend towards a lower pneumonia rate in patients treated with sucralfate, although this did not quite reach statistical significance.[60] The other study compared sucralfate to antacids in ICU patients, and showed a statistically significant reduction of pneumonias in the sucralfate-treated group (Fig. 4).[61] Further investigation suggested that the mechanisms for this reduction included both preservation of the gastric acidity and a direct antibacterial action of sucralfate.[62] These studies have been carried out in patients receiving mechanical ventilation in intensive

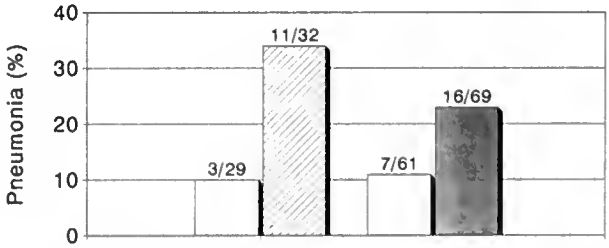

Fig. 4. In the study by Tryba (left panel),[61] nosocomial pneumonia occurred significantly less frequently in critically ill patients who received sucralfate ☐ than in those who received antacids ▨. In the study by Driks et al (right panel),[60] pneumonia occurred less frequently in critically ill patients receiving sucralfate than in those receiving antacids or H-2 blockers ☐, but the difference was not statistically significant.

care units, but no studies have been performed specifically in patients with ARDS.

SDD has been studied in several centers in Europe, primarily in the Netherlands, Germany, and the United Kingdom. The rationale for this therapy is that aerobic organisms colonize the oropharynx and gastrointestinal tract prior to colonization of the trachea and lung and prior to development of a nosocomial pneumonia. The normal flora of the upper airway and gut consists primarily of anaerobic organisms, which are helpful in protecting against colonization with other pathogenic organisms. Thus, the strategy is aimed at preventing aerobic colonization of the upper airway and gut while allowing normal anaerobic organisms to remain. The regimen includes administration of an intravenous antibiotic directed at gram-positive organisms for an initial few days while simultaneously beginning a prophylactic antibiotic combination consisting of tobramycin, polymyxin E, and amphotericin administered topically to the oropharynx and orally to the stomach. Several studies have been performed using this strategy in a wide variety of patient populations;[63-65] no studies have been performed in a study population consisting only of patients with ARDS. Few of the studies have concurrent randomized controls; most have used historical control groups. The overall results suggest that this regimen results in prevention of secondary or nosocomial infections, including a reduction in the incidence of pneumonia. However, to date, no evidence of a lowered mortality or shorter length of stay has been demonstrated. Importantly, no substantial increase in resistant organisms has been found.

Because previous trials of prophylactic antibiotics in ICU situations have resulted in an increase in resistant organisms,[66] this approach must be carefully studied and undertaken with caution. However, preliminary results in Europe warrant further consideration of this strategy and the performance of well designed, randomized clinical trials. Because of the reported high frequency of infections in patients with ARDS, and an associated increase in mortality,[1,32,33] this patient population seems to be ideal for further study employing this strategy.

Summary

In summary, the most widely proposed strategy to reduce mortality associated with ARDS consists of early identification of patients at high risk for developing the syndrome, followed by early therapeutic intervention. Three approaches to the prediction of ARDS development have been employed: early identification of physiologic derangements, measurement of blood-borne mediators or markers, and identification of clinical predispositions. Of these approaches, identification of clinical predispositions appears to be the most practical and successful. Clinical predispositions that allow appropriate study design and performance of clinical intervention trials have been identified. To date (1989), no intervention has been identified that prevents or ameliorates ARDS development. However, in the last 5 to 10 years, basic science and animal-model studies have identified several promising interventions. Several of these are suitable for early application in patients at high risk for developing ARDS. Some human studies are in progress and others are planned. Research on early identification of ARDS risks, complications of ARDS, mediators of acute lung injury and inhibitors of inflammatory mediators, and other agents that reverse physiologic derangements and prevent complications have all contributed to making the reduction of ARDS severity and mortality realistic and achievable goals.

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Full and Partial Ventilatory Support: The Significance of Ventilator Mode

Since the inception of intermittent mandatory ventilation (IMV) and synchronized IMV (SIMV), a tremendous debate has raged concerning the advantages and disadvantages of these modes as compared with the assist-control (AC) mode of mechanical ventilatory support. Recently in this forum, it has been suggested that the AC mode is superior to the IMV mode in providing full ventilatory support (FVS).[1] The primary reason for this assessment was the author's observation that patients ventilated in the IMV mode often demonstrate rapid, shallow patterns of ventilation between mechanically delivered breaths that have "cascaded into a great deal of extra work of breathing . . . ." The author further postulated that a change to the AC mode would result in a significant reduction or even elimination of this 'extra' work of breathing (WOB). In addition, he stated that "if one sets the [ventilator] rate high enough to totally support a patient on IMV, it becomes something other than IMV, because the ventilatory support is no longer intermittent; it is what Shapiro and Cane termed full ventilatory support" and reference was made to an editorial published in *Critical Care Medicine*.[2] These statements demonstrate a misunderstanding of several basic concepts concerning mechanical ventilatory support and a misinterpretation of the referenced literature.

First, Shapiro and Cane used two components to define FVS:[2] (1) they stated that it is a situation in which "the ventilator provides all energy required to maintain effective alveolar ventilation" and (2) they indicated that because "ventilator rates of 8 or greater in conjunction with tidal volumes of 12-15 mL/kg will result in P_{aCO_2} values < 45 torr in almost all patients . . . it is reasonable to arbitrarily define FVS as ventilator rates of 8/min or greater with tidal volumes of 12-15 mL/kg in conjunction with CMV [controlled mechanical ventilation], AMV [assisted mechanical ventilation], IMV, or SIMV." In this definition of FVS, there is no indication that IMV/SIMV rates must be set to ablate all spontaneous ventilatory efforts between mechanically delivered breaths; rather, the authors imply that the patient will receive adequate alveolar ventilation regardless of the presence or absence of spontaneous ventilatory efforts. Furthermore, care must be taken to avoid confusing the technical aspects of mechanical ventilation (ie, the mode of ventilation) with the physiologic level of ventilatory support (ie, FVS vs partial ventilatory support [PVS]). The mode of mechanical ventilatory support (CMV, AC, IMV, SIMV) remains the same regardless of the physiologic level of ventilatory support provided.

Second, the concept that the use of AC eliminates all WOB is incorrect. Marini et al[3,4] demonstrated that patient effort did not cease with the onset of gas delivery during assisted mechanical ventilation. They further showed that the patient component of the inspiratory work in the AC mode could be significant (30-50%) even under the most favorable conditions of minute ventilation ($\dot{V}_E$), demand-valve sensitivity, and inspiratory gas flowrates. Under less favorable conditions, the energy expended by the patient could exceed that required for mechanical inflation of the chest. These data clearly show that the AC mode can be associated with significant inspiratory work and that it does not necessarily provide complete ventilatory muscle rest.

Third (and most important, I believe) is the assumption that the WOB associated with the rapid and shallow ventilatory efforts sometimes noted with IMV/SIMV or the work associated with triggering the ventilator in the AC mode is in some way detrimental to the patient's overall cardiopulmonary homeostasis during FVS. This author is not aware of any scientific evidence indicating that the inspiratory WOB associated with any ventilatory mode providing FVS *is detrimental* to cardiopulmonary homeostasis. Even the documented increased WOB imposed by demand-valve systems is clinically important only with PVS or with spontaneous ventilation.[5]

Regarding the ongoing IMV/SIMV vs AC controversy, in 1983 Weisman et al[6] summarized the available information pertaining to the provision of FVS. The postulated advantages of IMV included a decreased risk of respiratory alkalosis, a decreased need for sedation, lower mean airway pressure ($\bar{P}_{aw}$), an improved distribution of ventilation, a more rapid withdrawal of ventilatory support, a decreased risk of respiratory muscle atrophy, and less interference with cardiovascular function. The purported disadvantages of IMV included an increased potential for hypercarbia, increased WOB and potential for respiratory muscle fatigue, prolongation of the weaning process (if the rate is not decreased appropriately), and a greater risk of hemodynamic instability during withdrawal of ventilatory support. Unfortunately, few clinical studies exist that adequately compare the IMV/SIMV mode with the AC mode during FVS in order to substantiate or dismiss these putative advantages and disadvantages. Recently, Groeger et al[7] performed a study utilizing a crossover protocol to compare AC and SIMV in terms of hemodynamic, metabolic, ventilatory, and oxygenation variables in patients with acute respiratory failure. The aspects of this study that compared AC and SIMV modes during FVS showed that spontaneous respiratory rate, P_{aCO_2}, mean arterial pressure, cardiac output (C.O.), oxygen delivery, and pulmonary artery occlusion pressure were all consistently higher in the SIMV group; pH and $\bar{P}_{aw}$ were consistently higher in the AC group. Oxygen consumption, carbon dioxide production and resting energy expenditure were similar in both groups. Unfortunately, as pointed out in the accompanying editorial by Kirby,[8] the apparent bias of the authors toward AC caused them either to fail to recognize or to refuse to acknowledge that their findings appear to favor SIMV in terms of the maintenance of overall hemodynamic and metabolic homeostasis.

However, despite these *statistically* significant data that suggest the superiority of SIMV over AC for the provision of FVS, the question remains: Do *clinically* significant differences really exist between the two modes during FVS? Considering the ongoing clinical success of both methods, the answer of most knowledgeable and rational clinicians must be: "No, probably not." Thus, we return to the real points raised by Shapiro and Cane.[2,9] First, it is primarily personal preference that determines the desirability of using IMV/SIMV or AC for the delivery of FVS. Second, because the AC mode cannot be used to provide PVS and because there is no demonstrable advantage in using AC (as opposed to SIMV/IMV) for the delivery of FVS, IMV/SIMV is the preferred mode of ventilation when PVS is planned. Newer modes of ventilatory support, such as pressure support ventilation (PSV), require further investigation to determine their role in the provision of PVS. Third, appropriately used PVS should enable the patient to assume an amount of WOB that is not detrimental to cardiopulmonary homeostasis and should provide the patient with some of the benefits of spontaneous ventilation. Therefore, the primary question is not which mode to use for FVS, but whether *full* or *partial* ventilatory support will optimize the specific clinical situation.

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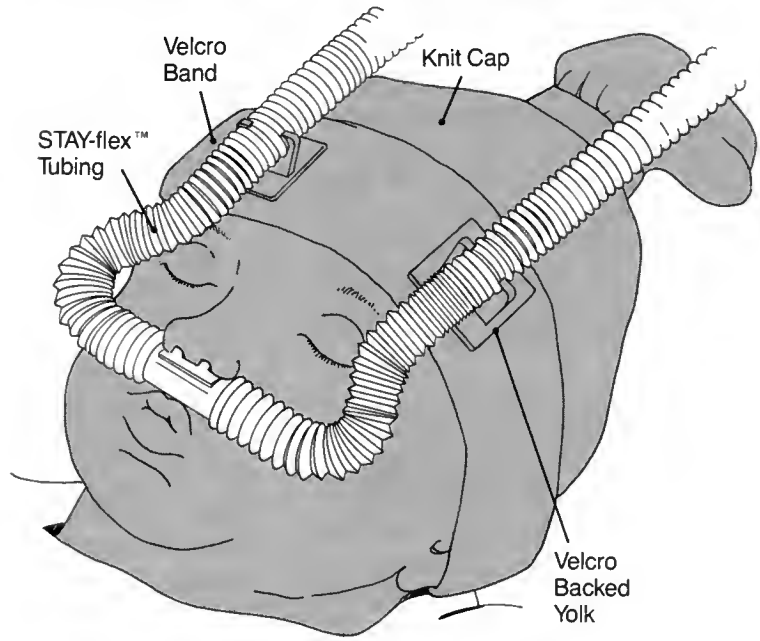

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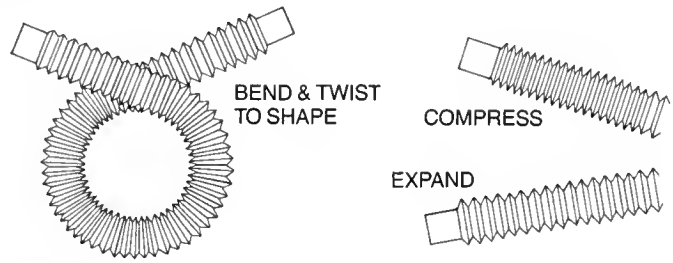

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Doctors: The Biography of Medicine, by Sherwin B Nuland MD. Paperback, 519 pages, illustrated. New York: Vintage Books (a division of Random House Inc), 1989. $11.95.

Doctors: The Biography of Medicine documents the evolution of modern medicine by presenting, in chronological order, the stories of 15 major breakthroughs in the study of disease. All but two of the chapters chronicle the lives of particular people whose accomplishments reflect the state of medical knowledge during that era.

Hippocrates, William Harvey, and Joseph Lister represent the better-known pioneers, but the contributions of physicians not as well known are also included. The story of Helen Taussig, for instance, illuminates the origins of pediatric cardiology and cardiac surgery. Taussig's story also demonstrates some of the difficulties faced by early female physicians in this country.

The chapters that deal with the invention of the stethoscope and the discovery of general anesthesia should be of particular interest to respiratory therapists. Taken in historic context, the technologies associated with respiratory care seem to be very recent developments compared to the origins of surgical care.

Dr Nuland describes the people and their culture so vividly that the reader can share the thrilling jolt of discovery. The author demonstrates the link between medical discovery and other events. The point is made, for example, that medical science always blossoms during wars. Dr Nuland is able to conjure up the horrors of 19th century hospitals with his highly evocative descriptions. He also supplies a wealth of fascinating medical trivia.

The biographies are somewhat ironic in that some of the issues the early pioneers grappled with are still plaguing health care today: drug addiction among physicians, controversy over abortion, protests against animal experimentation.

The author is a professor of surgery and medical history at Yale. His bias as a surgeon is occasionally evident, although he is able to provide insight into the ancient antagonism between diagnosticians and surgeons.

His work is carefully documented, with an extensive index and bibliography. I found some of the narrative to be redundant and dry, but, more often, the author goes to the other extreme and lapses into the "purple prose" that he criticizes in others. At one point, he refers to a theory that "had been untimely ripped from the gestating womb of scientific investigation . . . [when] what was required was a normal full-term pregnancy of research."

Doctors: The Biography of Medicine offers some excellent insight into how history has shaped medicine as we know it today. Unfortunately, only the contributions of Western schools of thought are showcased. No mention is made of the rich medical heritage of the Orient. Nevertheless, a substantial amount of information is presented, and it can be fascinating reading for those of us who are interested in the development of the art of healing.

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QuitSmart Stop Smoking Kit, 4th ed, by Robert H Shipley PhD. Illustrated paperback book and 30-minute audiocassette tape, 96 pages. Durham NC: JB Press, 1990. $14.95 plus $2.00 shipping. QuitSmart, PO Box 4843, Duke Station, Durham NC 27706. (919) 493-5221.

As a psychologist and former smoker, Dr Shipley, Director of the Duke University Quit Smoking Clinic, is well qualified to present smoking cessation material. He has written many scientific articles, books, and chapters on the subject of smoking cessation and has developed smoking cessation programs for the U.S. Veterans Administration and for such companies as Frito-Lay, PepsiCo, and Johnson & Johnson.

The QuitSmart Stop Smoking Kit consists of the book, **QuitSmart: A Guide to Freedom From Cigarettes**, and the audiocassette tape, **QuitSmart Self Hypnosis**. The book is aimed at cigarette smokers who would like to quit smoking on their own, but would also be useful to others attending a smoking cessation clinic. This easy-to-read pocket-size paperback has many light-hearted cartoons on the difficulties of quitting smoking. Easy-to-use charts emphasize some of the more important messages. The book contains many of the essential components of a good smoking cessation program; it effectively deals with nicotine addiction, physical withdrawal, and the psychological aspects of quitting.

The first chapter of the book prepares the smoker for quitting and introduces the concept of brand switching to reduce the addiction to nicotine prior to quitting 'cold turkey.' Proper use of Nicorette gum (Lakeside

Pharmaceuticals, Cincinnati, Ohio) is recommended to lessen withdrawal symptoms and weight gain after quitting.

The second chapter deals with quitting, coping with cravings, and physical withdrawal. Relaxation techniques are also introduced in this chapter.

The third and final chapter focuses upon how to remain both a nonsmoker and a healthier person. Many reasons for the former smoker to stay quit are offered, including health benefits and the new sense of control. This chapter enlists the help of the self-hypnosis tape. The deep soothing voice of Robert Conroy takes one through head-to-toe relaxation exercise. The first 15-minute side is aimed at those attempting to quit, suggesting healthy attitudes and reinforcing the decision to quit; the second 15-minute side encourages healthy living. Listeners are encouraged to take time out to relax and to reward themselves as new nonsmokers. The tape is to be used when quitting and for as long as needed afterward.

Overall, the **QuitSmart Stop Smoking Kit** can be recommended to physicians, respiratory therapists, and nurses as a good tool to use when helping patients to quit smoking.

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March 1-2 in Champion, Pennsylvania. The South Central District of the Pennsylvania Society for Respiratory Care hosts the 10th Annual Respiratory Care Symposium at Seven Springs Mountain Ski Resort in Champion. Joseph Golish MD, David Walker RRT, Kevin Corkery RRT, Ulf Borg PhD, and Trudy McGraw RN JD are the featured speakers. Contact Sandra Smith, University of Pittsburgh at Johnstown. (814) 266-9661, ext 412.

March 15-16 in Newport, Rhode Island. The Rhode Island Society for Respiratory Care presents its 6th Annual Respiratory Care Symposium "The Newport Challenge—Sails On." Contact Jayne Matoian RRT, PO Box 9051, Providence RI 02940-9051. (401) 723-8496.

April 4-6 in Baton Rouge, Louisiana. The Louisiana Society for Respiratory Care presents its 20th Annual Educational Meeting at the Embassy Suites. Contact Jim Lanoha RRT, (504) 387-7080.

April 11-13 in Nashville, Tennessee. The Tennessee Society for Respiratory Care presents its annual educational seminar and exhibition "1990s—A New Frontier" at the Vanderbilt Plaza Hotel in Nashville. Contact Colleen Schabacker, Cardiopulmonary Department, Jesse Holman Jones Hospital, 509 Brown St, Springfield TN 37172, or call (615) 384-1569. For exhibitor information, contact Candace Partee, (615) 449-0500.

April 17-19 in Philadelphia, Pennsylvania. The Pennsylvania Society for Respiratory Care presents its annual conference and exhibition at the Adam's Mark Hotel. This year's theme is "Putting on the Ritz for Our 25th!" Both respiratory care and cardiovascular technology are highlighted. Contact Jill Bucholtz, (215) 526-3341, or Wendy Janor, (215) 456-8049.

April 18-20 in Des Moines, Iowa. The Iowa Society for Respiratory Care presents its 1990 Iowa Lung Conference at the Hotel Jort Des Moines. Contact Mike Wheeler, (515) 283-6207.

April 18-20 in Rancho Mirage, California. The California Society for Respiratory Care Chapters 1, 2, 3, and 4 co-host their 9th Annual "Bright Horizons in Respiratory Care Symposium" at the Annenberg Center for Health Sciences on the Eisenhower Medical Center Campus. Speakers include David Pierson MD, John Bach MD, Dean R Hess MEd RRT, Bud Spearman BS RRT, Michael Gurevitch MD RRT; topics include pressure ventilation, barotrauma, auto-PEEP, MDIs vs SVN in ventilator therapy, ventilator trigger sensitivity and WOB, and noninvasive mask ventilation, plus presentations on neonatology, pediatrics, and oxygen transport and assessment. For more information, call (800) 321-3690. In California (800) 621-7322, or fax (619) 773-4513.

OTHER MEETINGS

March 27-28 in Chapel Hill, North Carolina. The respiratory care departments of Duke University Medical Center, Durham County General Hospital, Durham Technical Community College, and North Carolina Memorial Hospitals host their 4th Annual "Cardiopulmonary Technology Update." Contact Susan Rinaldo RRT, (919) 681-3273, or Pat Lovell RRT, (919) 470-5360.

April 17-21 in Orlando, Florida. Orlando Regional Medical Center presents its 11th Annual "Critical Care Medicine Conference" at the Hilton Hotel, Lake Buena Vista, next to Disney World. Contact Continuing Medical Education at Orlando Regional Medical Center, 1-800-648-0450.

April 20-22 from Miami to Nassau. Another 3-day, 2-night cruise and "floating seminar" on sleep-related disorders. Deadline for reservations is March 16. Contact Dave Robbins, (305) 441-6819.

May 20-24 in Boston, Massachusetts. The American Lung Association, its medical section, the American Thoracic Society, and the International Union Against Tuberculosis and Lung Disease sponsor The World Conference on Lung Health. Contact Maureen O'Donnell, American Lung Association, 1740 Broadway, New York NY 10019-4374.

August 30-September 8 Hawaiian cruise aboard the SS Constitution. Dream Cruises' 4th Annual Cruise for Continuing Education presents "Each One, Teach One." Fly to Oahu for 2 days in Waikiki before boarding ship to four other ports and the islands of Hawaii, Maui, and Kauai. Prices start at $1,215 plus airfare from your gateway city. Write Dream Cruises, 10882 La Dona Ave, Garden Grove CA 92640. 800-462-3628. CA residents call (714) 636-2566.

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Call for Abstracts

RESPIRATORY CARE
OPEN FORUM
1990 AARC ANNUAL MEETING

The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present papers at the Open Forum during the AARC Annual Meeting in New Orleans, Louisiana, December 8-11, 1990. Accepted abstracts will be published in the November 1990 issue of RESPIRATORY CARE. Membership in the AARC is not necessary for participation.

Specifications

Please Read Carefully

An abstract may report (1) an **original study**, (2) the **evaluation of a method or device**, or (3) a **case or case series**. Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, health occupations education, or management of personnel and health-care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal.

The abstract will be the only evidence by which the reviewers will decide whether the author should be invited to present a paper at the Open Forum. Therefore, *the abstract must provide all important data, findings, and conclusions.* Give specific information. Do not write such general statements as "Results will be presented" or "Significance will be discussed."

Essential Content Elements

An **original study** abstract ***must*** include (1) Introduction: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results.

A **method/device evaluation** abstract ***must*** include (1) Introduction: identification of the method or device and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience ***or*** a notation of lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate.

A **case report** abstract ***must*** report a case that is uncommon or of exceptional teaching/learning value and must include: (1) case summary and (2) significance of case. Content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

Abstract Format and Typing Instructions

An optical scanner will be used to process abstracts. ***Do not use a dot-matrix printer.*** First line of abstract should be the title. Title should explain content. Type the abstract ***double-spaced*** on ***plain white bond paper***, on ***one page*** only (copier bond is excellent). Do not underline or boldface and insert only one letter space between sentences. Provide a 1-inch margin top and bottom, a 1/2 inch left margin, and an approximate 1/2 inch ragged right margin.

No identification of authors or institutions is to appear on the abstract sheet or in the abstract itself. Make the abstract all one paragraph. Data may be submitted in table form ***provided the table width is limited to 60 letter spaces*** (ie, letters or numbers plus necessary blank spaces = 60). ***No*** figures or illustrations are to be attached to the abstract.

Type all information required to complete the author information form on the other side of this page. A photocopy of good quality may be used.

Standard abbreviations may be employed without explanation. A new or infrequently used abbreviation should be preceded by the spelled-out term the first time it is used. Any recurring phrase or expression may be abbreviated if it is first explained.

Check the abstract for (1) errors in spelling, grammar, facts and figures; (2) clarity of language; (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed.

Questions about abstract preparation may be telephoned to the editorial staff of RESPIRATORY CARE at (214) 243-2272.

Deadlines

The mandatory Final Deadline is June 9 (postmark). Authors will be notified of acceptance or rejection ***by letter only***—to be mailed by August 15.

Authors may choose to submit abstracts early. Abstracts received by March 26 will be reviewed and the authors notified by May 1. Rejected abstracts will be accompanied by a written critique that should in many cases enable authors to revise their abstracts and resubmit them by the final deadline (June 9).

Mailing Instructions

Mail 1 copy of the abstract, 1 author information sheet, and a stamped, self-addressed postcard (for notice of receipt) to:

RESPIRATORY CARE
PO Box 29686
Dallas TX 75229

1990 OPEN FORUM
AUTHOR INFORMATION SHEET

Please type:

Abstract title

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Institution

Please include authors' credentials.

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Mail station, building, or room

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Mail, with abstract and stamped self-addressed postcard, to RESPIRATORY CARE Open Forum
PO Box 29686, Dallas TX 75229

Notices of competitions, scholarships, fellowships, examination dates, new education programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notice to RESPIRATORY CARE Notices Dept, Box 29686, Dallas TX 75229.

Notices

AARC SUMMER FORUM

The Registry Resort, Naples, Florida, July 13-15, 1990
The Westin, Vail, Colorado, July 11-13, 1991

AARC ANNUAL CONVENTION SITES & DATES

1990—New Orleans, Louisiana, December 8-11
1991—Atlanta, Georgia, December 7-10
1992—San Antonio, Texas, December 12-15
1993—Nashville, Tennessee, December 11-14
1994—Las Vegas, Nevada, December 12-15
1995—Orlando, Florida, December 2-5

THE NATIONAL BOARD FOR RESPIRATORY CARE

1989 Examination and Fee Schedule

CRTT Examination

EXAMINATION DATE:	MARCH 10, 1990
Applications Accepted Beginning:	November 1, 1989
Application Deadline:	January 1, 1990
EXAMINATION DATE:	JULY 21, 1990
Applications Accepted Beginning:	March 1, 1990
Application Deadline:	May 1, 1990
EXAMINATION DATE:	NOVEMBER 10, 1990
Applications Accepted Beginning:	July 1, 1990
Application Deadline:	September 1, 1990

CPFT Examination

EXAMINATION DATE:	JUNE 2, 1990
Applications Accepted Beginning:	December 1, 1989
Application Deadline:	April 1, 1990

RPFT Examination

EXAMINATION DATE:	DECEMBER 1, 1990
Applications Accepted Beginning:	July 1, 1990
Application Deadline:	September 1, 1990

RRT Examination

EXAMINATION DATE:	JUNE 2, 1990
Applications Accepted Beginning:	December 1, 1989
Application Deadline:	February 1, 1990
EXAMINATION DATE:	DECEMBER 1, 1990
Applications Accepted Beginning:	June 1, 1990
Application Deadline:	August 1, 1990

Fee Schedule

Entry Level CRTT—new applicant:	$ 75.00
Entry Level CRTT—reapplicant:	$ 50.00
Combined RRT Written and Clinical Simulation:	$175.00
Written Registry Only—new applicant:	$ 75.00
Written Registry Only—reapplicant:	$ 50.00
Clinical Simulation Only—new or reapplicant:	$100.00
Entry Level CPFT—new applicant:	$100.00
Entry Level CPFT—reapplicant:	$ 80.00
Advanced RPFT—new applicant:	$150.00
Advanced RPFT—reapplicant:	$130.00
CRTT Recredentialing:	$ 25.00
RRT Recredentialing:	
Written Registry Examination	$ 25.00
Clinical Simulation Examination	$ 65.00
CPFT Recredentialing:	$ 25.00
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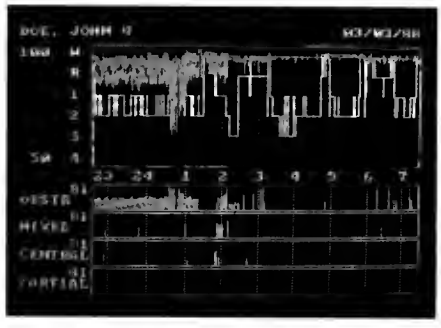

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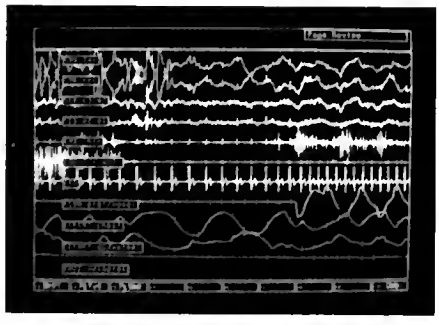

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RESPIRATORY CARE

Instructions for Authors and Typists

These Instructions are meant to guide authors and typists, including veterans in those roles, in the production of quality manuscripts. Perfection is not expected, but the well-prepared manuscript has the best chance for prompt review and early publication.

General Requirements

Submissions should (1) be related to respiratory care, (2) be planned for one of the publication categories below, and (3) be prepared as indicated in these Instructions. A letter accompanying the manuscript must specify the intended publication category, be signed by all the authors, and, when there are two or more authors, state that "We, the undersigned, have all participated in the work reported, read the accompanying manuscript, and approved its submission for publication."

Publication Categories

Research Article (Study): A report of an original investigation.
Evaluation of a Device/Method/Technique: A description and evaluation of an old or new device, method, technique, or modification.
Case Report: A report of a clinical case that is uncommon or of exceptional teaching value. The author(s) must have been associated with the case. A case-managing physician must be one of the authors or, if not an author, must supply a letter approving the manuscript.
Case Series: Like a Case Report but including a number of cases.
Review Article: A comprehensive, critical review of the literature and state of the art of a pertinent topic that has been the subject of 40 or more published research papers.
Overview: A critical review of a pertinent topic about which not enough research has been published to merit a Review Article.
Update: A report of subsequent developments in a topic that has been critically reviewed (not necessarily in this journal).
Point of View: A paper expressing the author's personal opinions on a pertinent topic.
Special Article: If a paper does not fit one of the foregoing categories but is pertinent, the editors may consider it as a Special Article.
Editorial: A paper that draws attention to a pertinent concern.
Letter: A signed communication about material published in this journal or on topics of interest or value to readers.
Blood Gas Corner: A brief, instructive case report (real or fictional) involving invasively or noninvasively obtained respiratory care blood data, followed by questions for readers—with answers and discussion.
PFT Corner: Like Blood Gas Corner but involving pulmonary function testing.
Test Your Radiologic Skill: Like Blood Gas Corner and PFT Corner but involving pulmonary-medicine radiography and including one or two 4 × 5 or 5 × 7 inch prints of radiographs. The case must be real.
Review of Book, Film, Tape, or Software: Anyone interested in writing a review can discuss it with an editor.

Editorial Consultation and Author's & Typist's Kit

To discuss a writing project, write to RESPIRATORY CARE, PO Box 29686, Dallas TX 75229 or call 214/243-2272.

Authors are urged to obtain the RESPIRATORY CARE Author's & Typist's Kit. The Kit provides *authors* with specific guidance about writing a research paper, writing a case report, converting to and from SI units, and in-house manuscript review. *Typists* can use the Kit's Model Manuscript, a list of journal name abbreviations, and a copy of these Instructions. The Kit is free from the Journal office.

Preparing the Manuscript

General Concerns—*Typist*

- Double-space ALL lines, including those in references, figure legends, and tables. Do not justify right margins.
- Number pages in upper right corner and leave margins of 1½" or more on all four sides of the page.
- For research articles, follow format of Model Manuscript, Respir Care 1984;29:182 (Feb 1984).
- Meticulously follow instructions for typing references.

General Concerns—*Author:*

- Structure manuscript as specified hereafter.
- Provide all requested information on title page as specified hereafter.
- Proofread manuscript for completeness, clarity, grammar, spelling; be sure all references, figures, and tables are cited in the text.
- Consider having paper reviewed in-house before submission.
- Have all co-authors proofread and approve manuscript and sign submission letter.

Manuscript Structure

Most kinds of papers have standard parts in a standard order. However, papers can vary individually, and not every paper will have all the parts listed here.

Research Article: Title page, abstract page, continuous text (Introduction, Materials & Methods, Results, Discussion), Product Sources page, Acknowledgments page, references, tables, figure legends. Please consult "Writing a Research Paper," Respir Care 1985;30:1057 (Dec 1985) and Model Manuscript, Respir Care 1984;29:182 (Feb 1984).
Evaluation of Device/Method/Technique: Title page, abstract page, continuous text (Introduction, Description of Device/Method/Technique, Methods of Evaluation, Results of Evaluation, Discussion), Product Sources page, Acknowledgments page, references, tables, figure legends.
Case Report or Case Series: Title page, abstract page, continuous text (Introduction, Case Summary, Discussion), Acknowledgments page, references, tables, figure legends. Also see "How To Write a Better Case Report," Respir Care 1982;27:29 (Jan 1982).
Review Article: Title page, Table of Contents page, continuous text (Introduction, History, Review of Literature, State of the Art, Discussion, Summary), references. May include figures & tables. No abstract. Table of Contents optional. Other formats may be appropriate.
Overview, Update, Point of View, or Special Article: Title page, text (introduction, message), references, tables, figure legends. No abstract.
Letter: Title page (provide a title), text, writer's name & affiliation, references. Tables & figures may be included. Double-space everything. Write "For Publication" on title page.

Structure: Important Details

Title Page: List title of paper, all authors' full names, degrees, credential letters, professional positions, and affiliations. List correspondence address, telephone number, and reprint address if desired. Name sources of grants or other support. Identify any author's consulting or commercial relationships that pertain to the paper's topic.

Abstract Page: Number this Page 1. List paper's title but omit authors' names. Abstract should be 200 words or less and must be informative, briefly specifying main points of paper, such as methods, results, and conclusions drawn.

Statistical Analysis: In research articles, identify statistical tests and chosen level of significance in the Methods section. In Results section, report actual P values.

Figures (illustrations): All photographs, diagrams, & graphs must be numbered as Figure 1, Figure 2, etc, according to the order in which each is first mentioned in the text. Photographs must be glossy prints 5 × 7 to 8 × 10 inches and should be black & white unless color is essential. Letters and numerals must be neat and large enough to remain legible if figure is reduced in size for publication. Final figures must be of professional quality, but 'rough' sketches may accompany the submitted manuscript, with final figures to be prepared after review. Identify each figure on back with a stick-on label showing figure number and arrow indicating top; omit author's name. Cover label with clear tape so ink will not smudge other prints. Supply three sets of unmounted figures. If figure has been published before, include copyright-holder's written permission to use it.

Figure Legends: List figure legends on a separate page, not on figures. If a figure has been published before, list the source in the legend.

Tables: Type each table on a separate page. Avoid more than 8 columns across. Continue a deep table on following pages. Give each table a number and descriptive title, placed above the table. Double-space ALL lines in tables, including column headings and footnotes.

Drugs: Brand names may be given, but always also show generic names.

Units of Measurement: In addition to conventional units of measure, show SI values and units in brackets after conventional expressions: ie, "PEEP, 10 cm H_2O [0.981 kPa]." For conversion to SI, see RESPIRATORY CARE 1988;33:861-873 (Oct 1988).

Commercial Products: If three or fewer commercial products are named in the text, list the manufacturer's name and location in parentheses the first time each is mentioned. If four or more products are named, do not list manufacturers in the text; instead, name the products and manufacturers in a Products Sources list at the end of the text. Provide model numbers when available.

Abbreviations: Use an abbreviation only if the term occurs several times in the paper. Write out the full term the first time it appears, followed by the abbreviation in parentheses. Thereafter, employ the abbreviation alone. Never use an abbreviation without defining it. Do not create new abbreviations unless absolutely necessary.

References:

- Use references to support statements of fact, indicate sources of information, or guide readers to further pertinent literature.
- Cite only published works—or works accepted for publication. When listing an accepted but still unpublished work, designate the accepting journal's name, followed by "(in press)."
- In the text, cite references by superscript numerals (half space above text), not in parentheses. The first reference cited in the text is number 1, the next is number 2, etc.
- In the reference list, place the cited works in numerical order.
- For the reference list, obtain author names, article and book titles, dates, volume and page numbers from the *original* cited articles and books, not from secondary sources such as other articles' reference lists, which often are inaccurate.
- Type references in medical-journal style. Examples appear at the end of these Instructions. Abbreviate journal names as in *Index Medicus*. A list of many journal-name abbreviations was published in Respir Care 1988;33:1050 (Nov 1988).
- DOUBLE-SPACE the lines of references.
- List ALL authors' names. Do not use "et al" to substitute for names.
- Identify abstracts, editorials, and letters as such. See examples.

Personal Communications, Unpublished Papers, and Unpublished Observations: List unpublished items in parentheses in the text, not in the reference list.

Examples of How To Type References

Notes: Although the examples here are printed with single-spaced lines, please double-space references in manuscripts. Also, note that words in article and book titles are not capitalized—except proper names.

Standard Journal Article:

1. Shepherd KE, Johnson DC. Bronchodilator testing: An analysis of paradoxical responses. Respir Care 1988;33:667-671.

Corporate Author Journal Article:

2. American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33:1044-1046.

Article in Journal Supplement:

(Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to allow retrieval.)

3. Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986;89(3, suppl):139s-143s.

Abstract in Journal:

(Abstracts are not strong references; when possible, full papers should be cited. When cited, abstracts should be identified as such.)

4. Lippard DL, Myers TF, Kahn SE. Accuracy of pulse oximetry in severely hypoxic infants (abstract). Respir Care 1988;33:886.

Editorial in Journal:

5. Rochester DF. Does respiratory muscle rest relieve fatigue or incipent fatigue? (editorial). Am Rev Respir Dis 1988;138:516-517.

Letter in Journal:

6. Smith DE, Herd D, Gazzard BG. Reversible bronchoconstriction with nebulised pentamidine (letter). Lancet 1988;2:905.

Personal Author Book:

7. Nunn JF. Applied respiratory physiology. New York: Appleton-Century-Crofts, 1969.

Note: To specify pages cited in a book, place a colon after the year and then list the page(s). Examples: 1969:85 (one page), 1963:85-95 (series of contiguous pages), 1963:85,95 (separated pages).

Corporate Author Book:

8. American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group, 1977.

Book with Editor, Compiler, or Chairman as 'Author':

9. Guenter CA, Welch MH, eds. Pulmonary medicine. Philadelphia: JB Lippincott, 1977.

Chapter in Book:

10. Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, eds. Pulmonary medicine. Philadelphia: JB Lippincott, 1977:171-223.

Submitting the Manuscript

After preparing the manuscript according to these Instructions, perform a final proofreading and check for accuracy and completeness. Then mail three copies of the manuscript and three sets of figures to RESPIRATORY CARE, PO Box 29686, Dallas TX 75229 (or Federal Express to RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229). Manuscript copy on IBM-compatible or Macintosh disks in addition to the requisite three hard copies will facilitate processing (Macintosh preferred). Enclose a letter as specified under **General Requirements** at the beginning of these Instructions. Do not submit material that has been published or is being considered elsewhere.

Author's Checklist

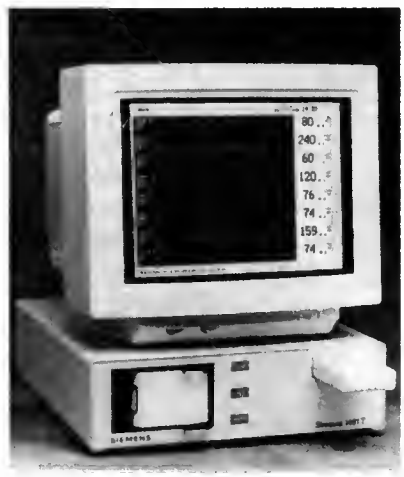
1. Is paper for a listed publication category?
2. Does cover letter meet specifications?
3. Is title page complete?
4. Are all pages double-spaced and numbered?
5. Are all references, figures, and tables cited in the text?
6. Are references typed in requested style?
7. Have SI values been provided?
8. Has all arithmetic been checked?
9. Has manuscript been proofread by all authors?

News releases about new products and services will be considered for publication in this section. There is no charge for these listings. Send descriptive release and glossy black and white photographs to RESPIRATORY CARE Journal, New Products and Services Dept, Box 29686, Dallas TX 75229.

New Products & Services

EDUCATIONAL RESOURCE PROGRAM. The Allen & Hanburys Respiratory Institute is a new program designed to provide information and to support projects that will promote an understanding of respiratory illnesses and their treatment among patients, the public, and medical professionals; first year emphasis is on asthma. A bimonthly newsletter, AIR CURRENTS, is available at no cost from the Institute; the newsletter includes current information on asthma causes and treatment, practical ways of dealing with the disease, guest columns by medical authorities, patient insight, discount offers on books, and information on items of interest to patients. AIR CURRENTS, designed to be of interest to asthmatics of all ages, has been launched with an initial circulation of 30,000. Allen & Hanburys Respiratory Institute, Dept RC, 301 E 57th St, New York NY 10022. (212) 715-1585 or (212) 593-6346.

DISPOSABLE MANUAL RESUSCITATOR. The Disposable Manual Resuscitator (DMR) is designed for single-patient use. The DMR is a disposable version of the Puritan Manual Resuscitator (PMR 2); it utilizes the reusable PEEP accessory and a disposable version of the nonrebreathing valve and bag-fill valve of the PMR 2. Additional features of the DMR, according to the manufacturer, are convenient storage (oxygen accumulator collapses to one third its original size), reduced operator fatigue (compliant, textured natural rubber bag), and added safety (kink-resistant oxygen tubing). Puritan-Bennett, Dept RC, 10800 Pflumm Rd, Lenexa KS 66215. (913) 469-5400.



CELLULAR TELEMETRY SYSTEM. According to the manufacturer, the SIRECUST 1481T telemetry system incorporates advanced digital transmission technology to help eliminate crosstalk and reduce signal interference at the viewing/recording station; and because any transmitter can be instantly tuned to a desired channel, the new system eliminates the need to keep spare transmitters. Additional features of the system include: multilead transmission, small water- and shockproof transmitter, space-saver viewing station with 14-inch color display, paper-saver mode that prints only what is needed for documentation (false-alarm paper waste is eliminated), 'soft-touch' 3-D controls, and pacer detection capability. Siemens Medical Systems Inc, Electromedical Div, Dept RC, 186 Wood Ave S, Iselin NJ 08830. (201) 321-4715.

OXYGEN CONSERVER. The OXYMATIC conserver is designed to extend the usable life of portable oxygen sources while the patient is away from his or her primary oxygen source, providing maximum mobility at minimal cost. According to the manufacturer, oxygen systems last an average of 7 times longer with the OXYMATIC conserver—a single 'D' cylinder (in conjunction with the OXYMATIC) can provide over 18 hours of ambulation to a patient on 2 L/min of O_2. CHAD Therapeutics Inc, Dept RC, 21630 Lassen St, Chatsworth CA 91311. (818) 882-0883 or (800) 423-8870.

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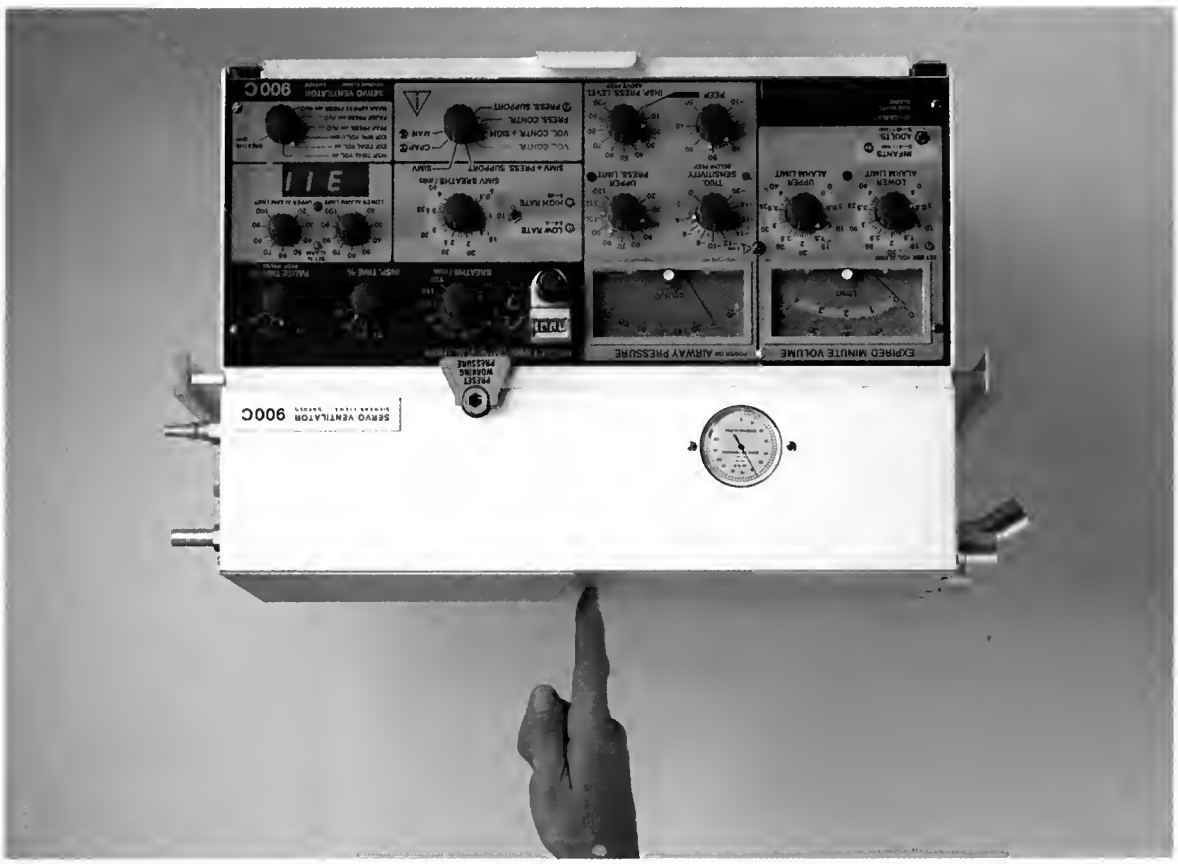

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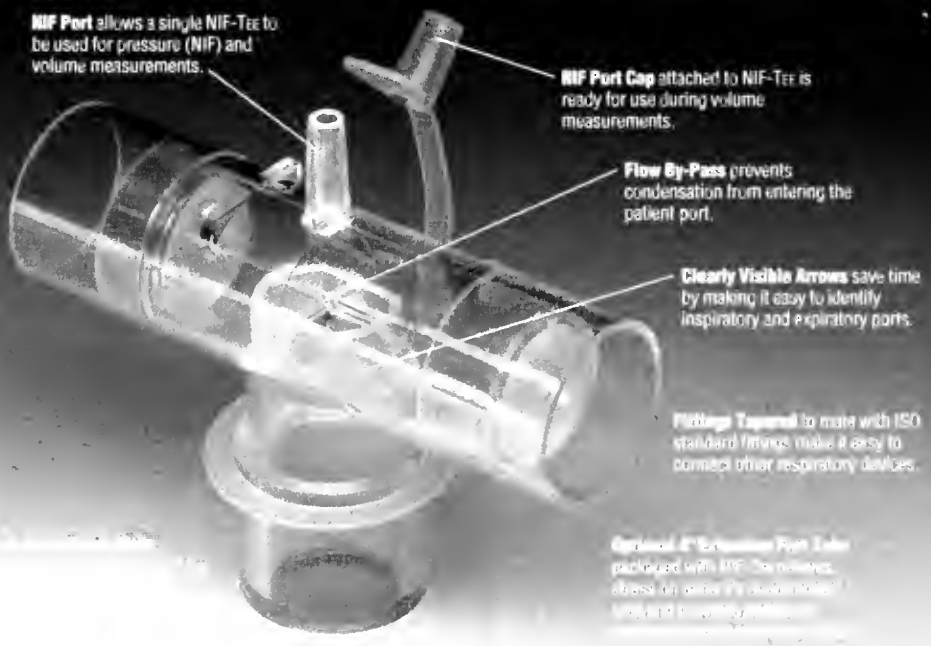

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